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THE PRACTICAL ANATOMICAL AND SURGICAL CONSIDERATIONS FOR EXPOSURE OF THE FACIAL NERVE.*

FRANK D. LATHROP, M.D.,
Boston, Mass.

I. – INTRODUCTION.

During the last war it was my privilege to be responsible for the care of a large number of casualties exhibiting complete peripheral facial paralysis secondary to battle wounds. Frequently these wounds involved the facial nerve not only in its course through the temporal bone but also in its distribution through the soft tissue of the face. In an effort to obtain information of a practical nature that would better enable me to deal with the injury to the nerve in such cases, considerable library research was carried out, which revealed that there was a paucity in the English literature of descriptions of the applied surgical anatomy of the facial nerve and surgical techniques necessary for its exposure at any point from the geniculate ganglion to the terminal branches. It is the object of this discussion to integrate the knowledge thus gained with that obtained through surgical experience with more than 100 cases of injury to the facial nerve.

It has long been a maxim of Dr. Lahey, when performing thyroid surgery, to isolate and expose the inferior laryngeal nerve in order to prevent paralysis of a vocal cord. While similar exposure of the facial nerve is not feasible during the

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course of a routine mastoidectomy, this principle can be applied with respect to the anatomical landmarks which determine its location. This maxim is directly applicable, however, when surgical procedures directed toward the parotid gland or soft tissues of the face are contemplated. Observance of this precept will not only insure that the facial nerve will not be unduly traumatized, but also will facilitate its exposure should the necessity arise.

II. – SURGICAL ANATOMY.

A. — The Temporal Bone.

In order to avoid injuring the facial nerve during the accomplishment of an adequately performed mastoidectomy, the primary prerequisite is a thorough knowledge of the exact surgical anatomy of this nerve in its course through the temporal bone. This applies equally well whether the surgical procedure employed be a simple or a radical mastoidectomy; furthermore, in the event that the operation to be performed is intended to expose the facial nerve because of the presence of an existing facial palsy, this prerequisite then becomes absolute if the lesion causing the facial paralysis is to be dealt with satisfactorily.

The best description of the surgical anatomy of the facial nerve with reference to temporal bone surgery is that presented by Sullivan, in 1938. I cannot improve upon the account of the applied anatomy he so ably set forth, and I desire only to re-emphasize its salient features. I also reiterate his opinion, proven in other fields of surgery, that it is not possible to avoid a structure if adequate surgery is to be done "if we do not know how and where to look for it."

In its course through the temporal bone the facial nerve is in intimate relationship to structures which can readily be recognized and utilized to depict its exact anatomical position. It is necessary that these landmarks be distinctly delineated during a mastoidectomy in order that the facial nerve may be visualized mentally and removal of the pathologic process within the mastoid expedited without injury to the nerve. The

facial nerve bears an undeviating relationship to the horizontal semicircular canal. This canal is readily recognized by otologists thoroughly trained and skilled in temporal bone surgery as a smooth, white, bony prominence situated in the anterior inferior aspect of the floor of the mastoid antrum which stands out in relief as compared to the adjacent diploic or

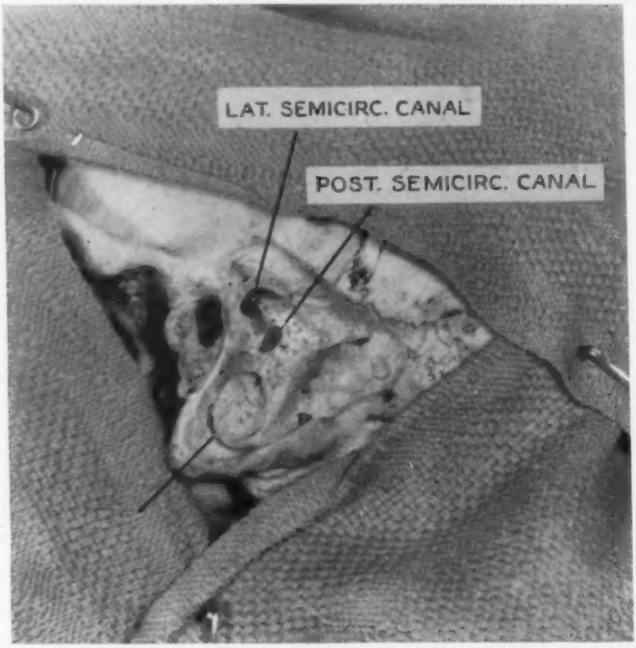

Fig. 1. Simple mastoidectomy; landmarks necessary for location of vertical segment of facial nerve are delineated.

cellular bone of the mastoid process. The horizontal semicircular canal marks the superior limit of the vertical portion of the facial nerve (see Fig. 1). At this level, the Fallopian canal is in intimate relationship to the anteroinferior aspect of the posterior end of the horizontal canal and is situated slightly medial to the eminence of the canal.

Immediately lateral to the Fallopian canal on the floor of the aditus ad antrum is the fossa incudis in which the tip of the short process of the incus rests. While not an important surgical landmark to the course of the facial nerve, it occasionally is of aid when the horizontal semicircular canal has been obscured or destroyed by trauma or disease. Surgical maneuvers medial to this point must be consummated with care.

The digastric groove, created by the posterior belly of the digastric muscle on the medial aspect of the tip of the mastoid

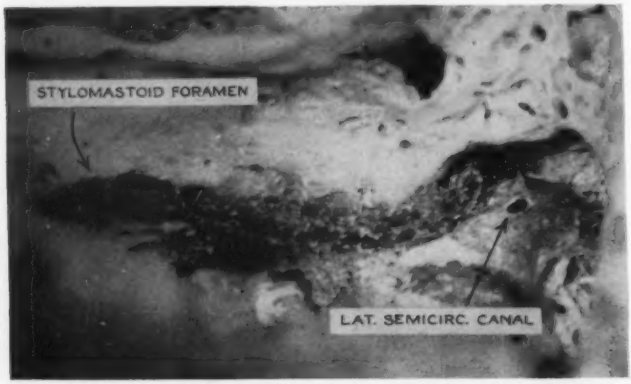

Fig. 2. Level of facial nerve between horizontal semicircular canal and stylomastoid foramen.

process, is represented as a gently concave ridge in the inferior portion of a mastoid cavity from which all tip cells have been removed and the plate of the sigmoid sinus clearly defined from the knee to the vicinity of the jugular bulb. The digastric ridge runs anterior and lateral to the sigmoid sinus to blend with the skeletonized posterior bony wall of the external auditory canal. Where the digastric ridge and posterior bony canal wall meet marks the stylomastoid foramen and denotes the inferior level of the facial nerve (see Fig. 1). Thus, the exact position of the vertical segment of the facial nerve may be determined by visually connecting the eminence of the

horizontal semicircular canal with the point where the digastric ridge and osseous external auditory canal blend. Surgical procedures necessary to eradicate a pathologic process medial or lateral to this line of demarcation may be accomplished without fear of traumatizing the facial nerve if the line of demarcation is respected (see Fig. 2).

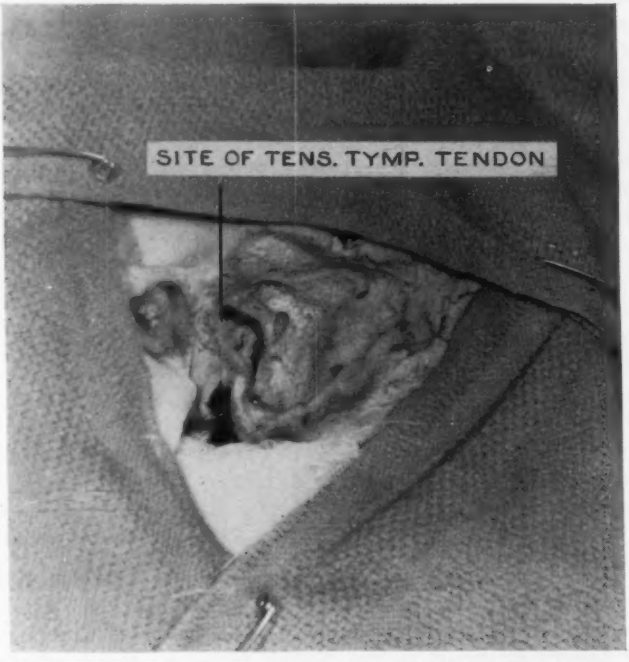

Fig. 3. Facial nerve exposed to show relationship to tendon of tensor tympani muscle.

The anterior extension of the intratympanic portion of the facial nerve is approximately represented by the point at which the tendon of the tensor tympani muscle is given off from the processus cochleariformis (see Fig. 3). While not an exact surgical landmark, it is a reliable guide to the posi-

tion of the nerve in this location during a radical mastoidectomy. In reality, the facial nerve runs medialward and slightly superior and anterior to this point to join the geniculate ganglion. For all practical purposes, however, the nerve is safe from surgical trauma while removing granulation tissue and necrotic bone from the tegmen tympani and expanding superior rim of the orifice of the Eustachian tube in this region so long as the cutting edge of a small curette is directed superior to the tendon of the tensor tympani muscle; furthermore, this latter structure is an invaluable guide to exposure of the geniculate ganglion. Posteriorly, the intratympanic segment terminates at the ampullary end of the horizontal semicircular canal. At this point it is slighty superior and lateral to the capitulum of the stapes and lies against the inferior and medial aspect of the anterior end of the horizontal semicircular canal.

The pyramidal portion of the facial nerve lies between the intratympanic and vertical segments. It is in intimate relationship with the inferior surface of the horizontal semicircular canal, lying slightly medial to the prominence of the canal. If the nerve is removed from the Fallopian canal in its pyramidal course it gives the impression that it lies in a groove on the inferior aspect of the horizontal canal. In this area the facial nerve lies close to the superoposterior wall of the middle ear and is susceptible to injury through injudicious use of the curette when removing granulation tissue or the annulus in this area (see Fig. 4).

This description of the surgical anatomy of the facial nerve is readily applicable to operations directed toward the mastoid process and tympanic cavity of the temporal bone. Cognizance of the relationship of the facial nerve to the landmarks described will permit such surgical procedures to be carried out without injury to the facial nerve and, when necessary, expedite exposure of the nerve at any point from the geniculate ganglion to the stylomastoid foramen. It must be emphasized, however, that merely a thorough knowledge of the surgical anatomy of the facial nerve is no substitute for adequate training and experience.

B. — Neck and Face.

Distal to the stylomastoid foramen, the facial nerve traverses the soft tissue of the neck and face to innervate the muscles for facial expression. In its facial distribution, injury to the nerve may occur as a result of deep lacerations of the face or during the course of operations upon the parotid or

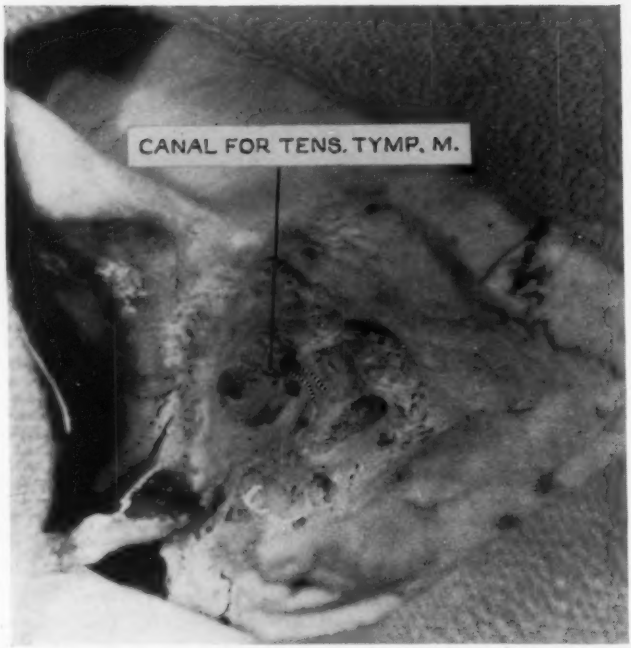

Fig. 4. Pyramidal segment of facial nerve outlined to demonstrate proximity to posterosuperior margin of tympanic cavity.

submaxillary glands, the temporomandibular joint and the soft tissue of the face for excision of tumors or scars. Since many otolaryngologists include such procedures in their field of practice, any discussion of the surgical anatomy of the facial nerve would be incomplete without a description of the applied surgical anatomy of the nerve in this region.

In its course through the soft tissue of the face the facial nerve is closely associated with structures which are of aid in determining its location. While the relationship of these landmarks to the facial nerve is not as exact as within the temporal bone, it is sufficient to permit the experienced surgeon to locate and expose the facial nerve in order that unnecessary

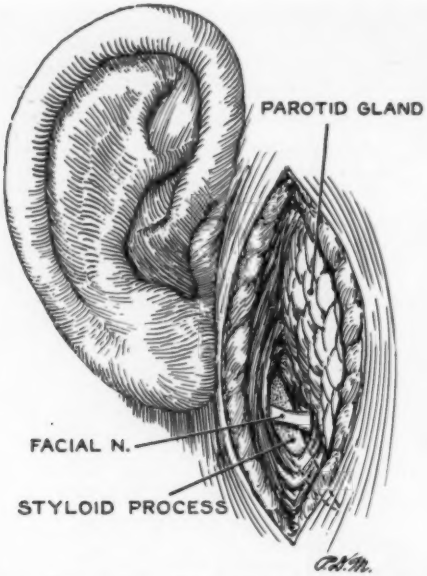

Fig. 5. Relationship of cervical trunk of facial nerve to styloid process.

trauma to the nerve may be prevented or reparative procedures instituted. The first landmark to be encountered after the facial nerve emerges from the skull is the styloid process. Emerging from the stylomastoid foramen, the nerve is directed anteroinferiorly and somewhat laterally to enter the posterior border of the parotid gland, and in this location lies either on or just lateral to the styloid process (see Fig. 5). Although it varies greatly in size, this structure is invariably

present and is a reliable guide to the position of the cervical portion of the facial nerve.

The facial nerve is intimately related to the parotid gland. The parotid gland is composed of two lobes connected by an isthmus and, contrary to common impression, the facial nerve does not penetrate the substance of the gland, but lies between the larger superficial lobe and the smaller deep lobe. This has been conclusively demonstrated by McCormack, Cauldwell and

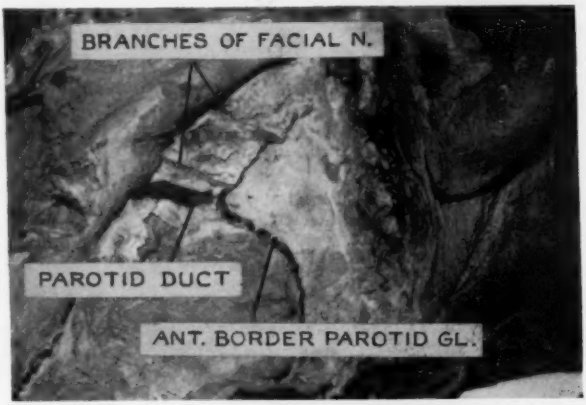

Fig. 6. Relationship of temporofacial division to anterior border of parotid gland and parotid duct.

Anson. Shortly after entering the parotid gland, the cervical trunk of the facial nerve regularly divides into two principal divisions, the temporofacial and cervicofacial portions, which lie on either side of the isthmus. The point at which this division occurs is of little surgical significance but is roughly located medially to the anterior border of the lobule of the external ear or two-thirds of the distance from the angle of the mandible to the temporomandibular joint and just posterior and medial to the ramus of the mandible.

The temporofacial and cervicofacial divisions continue to run forward and further divide between the superficial and deep lobes of the gland to appear at its anterior border. The

former is the larger and more important of the two divisions; its branches emerge from under cover of the superficial lobe at its anterior and superior margin in relationship to the zygomatic arch and parotid duct (see Fig. 6). This latter structure is an important landmark with respect to the surgical anatomy of the facial nerve distal to the parotid gland, as the zygomatic or buccal branch of the facial nerve runs transversely across the face just above the parotid duct and often crosses it. The inferiorly directed cervicofacial division emerges at the lower pole of the parotid gland, from which point the mandibular branch may be found running along the body of the mandible or just inferior to it.

Although considerable anatomical variation with respect to the relationship of these landmarks to the facial nerve may exist, the deviation is not sufficient to prevent them from being of definite value when location of the cervical trunk or the principal branches of the facial nerve distal to the stylomastoid foramen is necessary. The utilization of these landmarks will enable an experienced surgeon to "pick up" the facial nerve, either proximal or distal to the parotid gland, with a minimum of difficulty and will facilitate exposure of the nerve in its course between the lobes of the parotid gland.

III. – METHODS OF EXPOSURE.

A. — Exposure in the Temporal Bone.

Exploration of the facial nerve is indicated when facial palsy supervenes immediately following an apparently uneventful mastoidectomy. For this purpose the nerve may be "picked up" at several points in its course through the temporal bone if the landmarks previously described are utilized to locate its position in the area in which exposure is desired. From this point the facial nerve can be further exposed to include all or any part of its vertical, pyramidal or intratympanic segments. It is a prerequisite, of course, that appropriate surgical eradication of the pathologic process within the mastoid or tympanic cavity has been accomplished.

The sole exception to this requirement is when decompression of the facial nerve is undertaken for Bell's palsy.

The facial nerve in its vertical course through the mastoid is most easily "picked up" as it emerges from the stylomastoid foramen. The mastoid tip is removed in its entirety with gouge

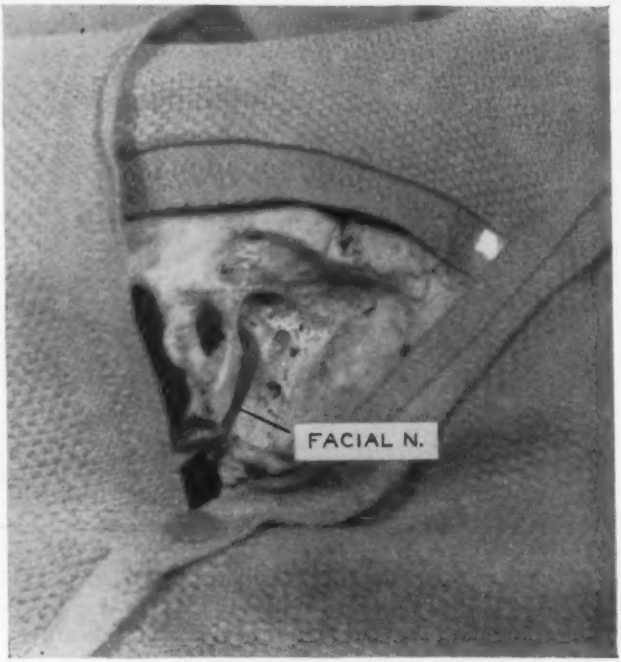

Fig. 7. Facial nerve exposed from stylomastoid foramen to horizontal semicircular canal after removal of mastoid tip.

and mallet or rongeurs to the level of the digastric groove, which is then followed anteriorly to the posterior rim of the stylomastoid foramen. The periosteum and fibrous tissue of this area resembles a small inverted funnel with the spout corresponding to the vertical portion of the facial nerve. The bone forming the posterior and lateral borders of the stylo-

mastoid foramen, as well as that covering the immediately adjacent Fallopian canal, is shaved down to the thickness of paper with gouge and mallet or an electrically driven burr parallel to the vertical course of the facial nerve. Removal of the remaining thin plate of bone exposes the nerve. This is most readily accomplished by inserting the tip of a small curette or dental pick underneath the edge of this thinned plate of bone at the stylomastoid foramen and lifting the instrument away from the facial nerve so as to remove the engaged portion of bone. Repetition of these maneuvers along the vertical course of the facial nerve will permit its exposure from the stylomastoid foramen to the horizontal semicircular canal (see Fig. 7).

The facial nerve may be exposed also in the stylomastoid foramen and the inferior end of the Fallopian canal from within the mastoid cavity. The digastric ridge is the keynote to success with this approach to the facial nerve. The anatomical tip of the mastoid process is not removed in this procedure, but it does necessitate careful removal of the cellular structure of the tip and exposure of the plate of the sigmoid sinus in the inferior aspect of the mastoid cavity in order that the digastric ridge may be sharply defined. The point at which the ridge meets the skeletonized posterior wall of the external auditory canal marks the inferior end of the Fallopian canal and stylomastoid foramen. The facial nerve may be exposed at this point from within the cavity of the mastoid by gentle removal of thin shavings of bone parallel to the vertical course of the nerve with a medium sized sharp curette or electric burr until the neurilemmal sheath is observed. Exposure of the facial nerve is then obtained by removing the overlying bone piecemeal with a small, sharp curette, the cutting edge of which is held parallel to the axis of the nerve (see Fig. 8). This method of approaching the facial nerve, while more difficult than that described above, is particularly adaptable to those cases of Bell's palsy in which decompression of the facial nerve is deemed advisable, since its employment causes a minimum of postoperative discomfort and disability.

Exposure of the pyramidal segment of the facial nerve may be accomplished by using the horizontal semicircular canal as a guide. Careful removal of the bone lying immediately anterior and inferior to the horizontal semicircular canal in the manner previously described will expose the nerve in this area. While a radical mastoidectomy is not absolutely necessary to

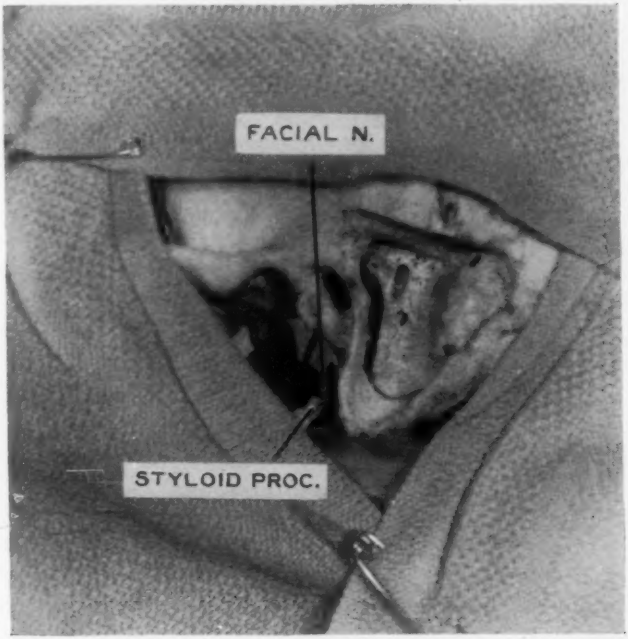

Fig. 8. Facial nerve exposed from stylomastoid foramen to horizontal semicircular canal without removal of mastoid tip.

permit adequate exposure of the pyramidal portion of the facial nerve in a well pneumatized temporal bone if the incus is removed, it is almost invariably a prerequisite in the event a nerve graft must be utilized to repair a defect of the nerve in this region (see Fig. 8).

The intratympanic segment of the facial nerve is accessible after a radical mastoidectomy has been performed. This is

the only portion of the facial nerve that is not buried deeply within the temporal bone. It may be recognized as a rounded elevation running horizontally across the upper limit of the tympanic cavity immediately inferior to the anterior end of the horizontal semicircular canal and surgical dome of the vestibule. The nerve in this location is covered by extremely thin and brittle bone, which facilitates its removal. Exposure

Fig. 9. Facial nerve exposed from stylomastoid foramen to vicinity of geniculate ganglion.

is best effected by engaging the cutting edge of a small curette at a point on its greatest convexity and, with sufficient pressure at a tangential plane, fracturing and removing a small fragment of bone. From this point the nerve may be completely uncovered by removing its bony covering piecemeal with the point of a small curette or dental searcher (see Fig. 9).

B. — Exposure in the Neck and Face.

The facial nerve may be isolated at several points distal to the stylomastoid foramen. Using this point as a focus for further exposure, the nerve may then be traced throughout its course in the soft tissue of the neck and face. The simplest way to accomplish this is to locate the facial nerve as it emerges from the stylomastoid foramen by removing the tip of the mastoid process as described in an earlier part of this paper. Thereafter, the cervical trunk of the facial nerve may be traced forward through the parotid gland and into its facial arborization by sharp and blunt dissection. A nerve stimulator may be utilized to identify and facilitate the exposure of the nerve as the dissection is carried forward.

An alternative method of locating the facial nerve proximal to the pes anserinus is that employed by Maxwell. An incision is made through the skin and subcutaneous tissue just anterior to the tragus, beginning just above its upper margin and extending downward several centimeters below the inferior border of the lobule of the ear. Closely hugging the anterior wall of the external auditory canal, the posterior surface of the parotid gland is freed by blunt dissection until the base of the styloid process is located. This is the keynote to this approach, since the styloid process maintains a close relationship to the facial nerve as it courses forward from the stylomastoid foramen. The dissection is carried along the lateral surface of the styloid process until the cervical trunk of the facial nerve is located (see Fig. 5). Once isolated, further exposure is accomplished as described in the previous paragraph.

The inframandibular branch of the facial nerve may be employed to locate the pes anserinus when operating on the parotid gland. Sistrunk isolated this branch, as it runs beneath the platysma muscle 1.5 cm. inferior to the angle of the mandible through a curved incision 2 cm. below the lower border of the mandible and ending 3 cm. above the mastoid tip and 4 cm. anterior to the angle of the mandible. The nerve is dissected up through the substance of the parotid gland to

the point at which the cervical trunk of the facial nerve divides into the temporofacial and cervicofacial divisions.

Adson combined this approach with one of his own to expose the facial nerve within the parotid gland. A vertical incision just anterior to the ear, beginning at the zygoma, is curved around the lobule to join Sistrunk's incision. After the skin and subcutaneous tissue are reflected from the superficial lobe of the parotid gland, the superior branches of the facial nerve are located as they cross the zygoma. These are dissected

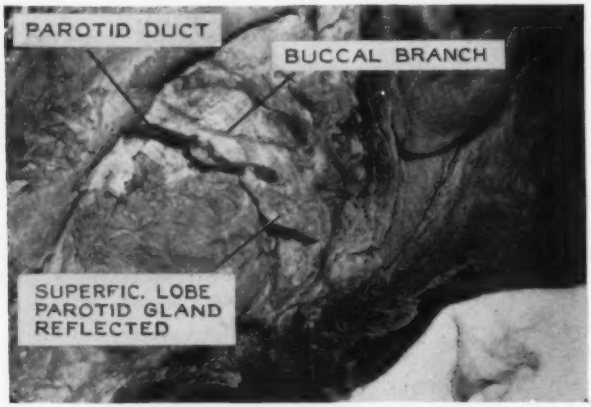

Fig. 10. Relationship of buccal branch of facial nerve to parotid duct.

through the gland to locate the pes anserinus. The cervicofacial division is then exposed, following the technique of Sistrunk. In this way the superficial lobe of the parotid gland may be elevated from the underlying facial nerve and removed without permanent injury to the nerve.

The buccal branch of the facial nerve distal to the parotid gland maintains a close relationship with the parotid duct. If the anterior border of the gland is exposed through an incision just anterior to the ear, beginning at the temporomandibular joint and curving slightly anteriorly to terminate just below the angle of the mandible, this branch of the facial

nerve may be located, as it runs slightly superior and parallel to the duct in the area just anterior to the border of the gland (see Fig. 10). From this point the anterior border of the superficial lobe of the parotid gland may be elevated superiorly and inferiorly to expose the remaining branches of the facial nerve. Keeping the branches of the nerve in constant view by retracting the superficial lobe of the gland, the dissection is carried posteriorly until the converging branches unite, to be continued proximalward as the cervical trunk. Transection of the isthmus of the gland parallel to the facial nerve permits removal of the superficial lobe of the parotid gland and visualization of the branches of the facial nerve throughout their course from the distal end of the cervical segment to the smaller branches in its facial distribution.

SUMMARY.

The surgical anatomy of the facial nerve within the temporal bone, neck and face has been described. Identification and application of the anatomical landmarks specified will accurately designate the course of the nerve within the temporal bone and, to a lesser degree, that of the facial nerve in the neck and face.

Methods for exposing the facial nerve in several locations have been given. Thorough familiarity with these methods will permit exposure of the facial nerve at any point from the geniculate ganglion to its distal branches and enable the surgeon to utilize the approach best adapted to the individual case.

While a thorough knowledge of the surgical anatomy of the facial nerve will permit the surgeon to visualize mentally its location, it does not necessarily follow that he is capable of its exposure. Adequate training and experience in surgery are prerequisites for such a surgical procedure and, in addition, the surgeon must be equipped mentally and physically to perform such surgery. The surgeon operating on such patients must be thoroughly conversant with the anatomy of this region and be blessed with an infinite amount of patience and perseverance.

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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose and Throat Society will be held in Charleston, S. C., Sept. 13-16, 1948. Headquarters will be the Francis Marion Hotel. The first two days will be devoted to ophthalmology and the last two to otolaryngology. Four outstanding men have been secured in each of the above specialties. For additional information, please write to Dr. Roderick Macdonald, Secretary, Rock Hill, S. C.

THE ARTICULATION AREA AND THE SOCIAL ADEQUACY INDEX FOR HEARING.*

HALLOWELL DAVIS, M.D.,
St. Louis, Mo.

The most important function of hearing for modern man is in auditory communication. What the deaf man wants most is to hear human speech, so that he may understand. The other functions, such as warning of danger, enjoyment of music, controlling the quality and loudness of his own speech, and locating and identifying miscellaneous sounds and signals, are secondary to the hearing of speech or closely related to it.

There is an obvious need for a quantitative measure of the social adequacy of hearing, not only for medicolegal purposes but also to assess the therapeutic success of remedial procedures such as the fenestration operation, auditory training, or the fitting of a hearing aid. The pure-tone audiometer gives a series of measures of hearing loss, in decibels as a function of frequency. It is excellent for medical diagnosis, but for the present purpose it is too analytic. There is no simple or fully satisfactory way of translating the pure-tone audiogram into a single index which will express the ability of a patient to hear speech. The best attempt to do this is the table and rules set forth for medicolegal purposes by the Consultants on Audiometers and Hearing Aids of the American Medical Association.[1] The continuing debate as to the accuracy and adequacy of these rules suggests, however, that they are not entirely satisfactory; and no attempt has been made to validate them experimentally. The chief difficulty lies in assigning the proper weights to the hearing losses at different frequencies.

A more direct approach to the problem was suggested in 1946 by Walsh and Silverman.[14] They proposed the direct

*Read at the Fifty-second Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., April 8, 1948.

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measurement of hearing loss for speech by using actual speech as the test material instead of pure tones. This was not new, but they pointed out that it is not enough to measure the hearing loss in decibels. We must also measure the ability of a patient to *discriminate* a variety of words and repeat them correctly. They proposed to measure directly the patient's "articulation score" at three intensity levels representative of the range of actual everyday intensities of speech, and to take as an index of social adequacy *the average overall percentage of (monosyllabic) words correctly understood.* As appropriate material for such a test they used the phonetically balanced (PB) word lists prepared by the Psycho-Acoustic Laboratory. These lists represent the various vowel and consonant sounds in the proportion in which they actually occur in average English speech. These lists have, in technical language, a "high face validity" as fair material with which to measure the ability to hear speech.

The present study undertakes to *1.* develop a convenient method for applying these principles systematically in practice, *2.* to consider numerical values on this scale for the "threshold of social adequacy," and *3.* to examine the properties and implications of the "social adequacy index" and some of its relations to other tests of hearing loss for speech and pure tones. In particular, its use in evaluation of the success of the fenestration operation and in predicting its probable benefits will be indicated.

"THE ARTICULATION AREA."

The basis of the social adequacy index is the relationship between *1.* the percentage of words correctly understood, which is known as the articulation score, and *2.* the *intensity level* at which the words reach the ear of the listener. The second quantity is measured physically in decibels relative to an arbitrary level (usually 0.0002 dyne/cm^2) by means of an appropriate measuring instrument known as a VU meter. The curve that expresses the relationship of articulation score to intensity is the "articulation curve."* A normal articulation

*For a general discussion of the articulation curve and of tests of hearing see Reference 6, Chapter 6.

curve is shown in Fig. 1 (heavy curve). This particular curve was determined experimentally at Central Institute for the Deaf, for 10 normal listeners, using a particular set of recordings of PB word lists and a high-fidelity electroacoustic playback and reproducing system. It agrees closely with similar curves determined at the Psycho-Acoustic Laboratory.[7]

The normal articulation curve shows that the most intelligible word in a PB list (of 50 words) is correctly heard at an intensity level of about 20 db (above 0.0002 dyne/cm^2). This level is 11 db above the average threshold intensity of pure tones in the speech range for the same listeners. The articulation curve rises at the rate of nearly 4 per cent per decibel increase in loudness until at 33 db the average normal listener repeats correctly 50 per cent of the words. This intensity (not the foot of the curve) is known as the "threshold" for the PB word list, according to the usual convention for defining the threshold of a word or sentence test.

Above this 50-per-cent correct level the curve rises more and more slowly, and all of the words are not correctly heard until an intensity of about 70 db (above 0.0002 dyne/cm^2) is reached. This level is about 50 db above the intensity at which the easiest words are recognized, and is about the level at which average conversational speech reaches the listener. Notice that if a PB word list is read to a normal listener at the level of *faint speech* (55 db re 0.0002 dyne/cm^2) frequently encountered in church or at lectures, he will miss, on the average, about 5 per cent of the words.

The coordinates on which the articulation curve is plotted in Fig. 1 are *intensity* (in decibels) as the abscissa and *percentage of PB words correct* or *percentage of PB words missed* as the ordinate. For some purposes it is convenient to use the traditional "articulation score" based on words correct, but for other purposes, notably for describing the hearing impairment of a patient, it is more convenient to use the right-hand scale based on words *missed*. This scale we shall call "discrimination loss." The two dimensions of auditory impairment then become *discrimination loss* (percentage of errors) and *hearing loss* (in decibels).

THE ARTICULATION AREA
ARTICULATION SCORE : Percent PB WORDS CORRECT
DISCRIMINATION LOSS : Percent PB WORDS MISSED
SOUND PRESSURE IN DECIBELS RE 0.0002 Dyne per Cm²
Discomfort begins
LOUD
AVERAGE
FAINT
ORDINARY SPEECH RANGE
NORMAL PB ARTICULATION CURVE : S A I • 98
Limit of Normal : S A I • 94
NORMAL THRESHOLDS
PB
No. 12 and 4C
Connected Discourse
No. 9
Detectability
Pure Tones in Speech Range
Two Social Adequacy Threshold Curves S A I • 33
Maximum Pure Conductive Loss : 50 db
SCALES OF HEARING LOSS FOR SPEECH
No. 12, 4C
C.D. No. 9
PB

"The Articulation Area" (see Fig. 1) is limited to the left by the acuity of hearing. To the right it is bounded by the threshold of tolerance. An average value for discomfort (for speech) is 120 db re 0.0002 dyne/cm^2.[11] Most listeners, whether normal or hard-of-hearing, will not accept greater intensities in ordinary situations, although some will go up to 130 or 135 db if necessary. Simple hearing loss (with a "flat" audiogram), such as may be produced by otosclerosis, moves the articulation curve bodily to the right and thus reduces the patient's articulation area.

The limit of normal hearing, corresponding to a loss for speech of 9 db, is indicated by the dashed articulation curve. (Nine decibels of hearing loss for speech is about three times the standard deviation (σ) of the normal threshold for Test No. 9. Falconer and Davis[8] found $\sigma = 2.6$ db: Breakey[3] found $\sigma = 3.13$ db.)

The scale of hearing loss for speech as measured by the PB word list is laid off in Fig. 1 along the 50-per-cent-missed abscissa.

The ordinary speech range is indicated by the dashed vertical lines. Loud speech close to the ear or something close to a shout at six feet has an intensity of about 80 db when measured in a free field. The scale of sound pressures in Fig. 1 refers to sound pressure measured under a receiver. This is not the same as 80 db measured in a free field because the head and ear when introduced into the free field modify the sound waves there. Resonances in the ear canal also modify the sensitivity of the ear. The net effect is that speech at

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Fig. 1.

The PB articulation curve and the thresholds for the various speech tests are based on receiver listening on the apparatus employed in Laboratory No. 2 of Central Institute for the Deaf, 1944 to 1948. The articulation curves apply to the PB word lists of the Psycho-Acoustic Laboratory as recorded at the Technisonic Studio (Central Institute for the Deaf) spoken by Rush Hughes. Slightly different shapes of curves and different values for thresholds may be found with other apparatus and other recordings.

The section of the normal PB articulation curve from 5 per cent to 55 per cent correct is straight with a slope of 50/13 (3.85 per cent per decibel). The slight "tail" to the left at the bottom leaves the base line at 19 db. The curve crosses the 50 per cent correct line at 33 db and the upper half is adequately defined by the following representative points: 36 db, 60 per cent; 40 db, 73 per cent; 46 db, 86 per cent; 56 db, 96 per cent; 70 db, 100 per cent.

SAI refers to the Social Adequacy Index tabulated in Fig. 2. It is the average percentage of PB words that would be correctly understood by the listener at the three speech levels indicated by the dashed lines.

80 db by free field measurement is heard as loudly and as well as speech at 85 db as measured under a receiver.* Since we are using the receiver scale, we plot "loud speech" at 85 db, instead of at 80. Similarly, we locate conversational speech at 70 db instead of at the traditional 65 db. "Faint speech," corresponding to church or a lecture, we place at 55 db. The difference between "faint" and "loud" speech is taken somewhat arbitrarily, but conveniently, as 30 db. This is within 2 db of the corresponding figure (28 db) adopted independently for a very similar purpose (the evaluation of hearing aids) by the Otological Research Unit of the British Medical Council.[5]

DISCRIMINATION LOSS.

Pure hearing loss for speech is illustrated by two articulation curves at the right, one solid (44 db hearing loss) and one dashed (50 db hearing loss). Notice that each of these curves is normal in shape and rises to a perfect articulation score (no words missed). Even the most difficult words are heard correctly if they are made loud enough, and for these hearing losses the words *can* be made loud enough without becoming uncomfortable.

The dashed curve at 50 db loss represents probably the greatest loss for speech that can be caused by pure conductive deafness. If words (or pure tones) are delivered to the ear, either through a receiver or in a free field, at 50 db above normal threshold they begin to be heard *by bone conduction* if the inner ear is normal. (The "shadow" audiogram 45 or 50 db below normal due to hearing by a normal ear on the opposite side of the head if masking is not used is a familiar and direct demonstration of this principle.) In a free field at this level the skull picks up acoustic energy directly and

*The difference between "free field" listening in our sound-treated room and receiver listening we determined experimentally for speech by Test No. 12. For tests on 10 normal ears, monaural listening, the difference in threshold was 4.75 db.[3] The value of 5 db is a round-number compromise between this value and the classical 7 db difference in the pure-tone thresholds (minimum audible field vs. minimum audible pressure) averaged over the frequency band 300-3000 c.p.s. The discrepancy is not critical because the difference between the two scales only enters our argument in locating the typical speech levels, and this location is admittedly arbitrary in any case. Hearing losses for speech are calculated by subtraction of a normal thresho'd value which has been determined for the particular test **and for the particular conditions of listening.**

transmits it to the inner ear without the assistance of the ossicles. (Further evidence of this maximum of conductive deafness at about 50 db hearing loss will be discussed elsewhere. The concept is of great importance for prognosis of the fenestration operation, because the operation can alleviate only conductive deafness.) If tests show a loss for speech greater than 50 db it is usually safe to conclude that there is a loss of sensitivity of the sense organ (nerve deafness) as well as conductive deafness.

Nerve deafness may cause hearing losses for all frequencies, and insofar as it affects them all *alike* it is indistinguishable, by simple air-conduction threshold tests, from conductive hearing loss (but *cf.* Békésy[2]). Usually, however, nerve deafness affects the high tones more than the low tones, and if severe the hearing loss for some high tones may be complete. In this case some words, usually those containing weak, high-pitched fricative consonants or sibilants, are never correctly heard, *no matter how loudly they are spoken.* The articulation curve rises, perhaps to 50 or 40 per cent discrimination loss, and then flattens out on a smooth plateau. (Often, if the intensity is increased to 120 db or higher, the curve actually falls again, due to distortion of loud, low tones in the ear of the listener or to distraction by the discomfort.) *High-tone nerve deafness thus shifts the entire articulation curve downward.*

It might be argued that in mixed deafness the curve is not shifted downward as a whole, but that the curve is shifted to the right and is also *truncated* by a lowering of the plateau. If the curve is truncated the angle between the rising portion and the plateau will be sharper than normal, as in the figure published by Walsh and Silverman.[14] As yet we do not have enough sufficiently reliable articulation scores obtained at just the right intensity to settle the question. The experimental difference is less than the standard error of measurement of a single PB test, however.

For simplicity, and subject to correction at a later date, we will assume, in deriving the social adequacy index, that the articulation curve always retains its normal shape, but that it may be shifted to the right by any "flat" hearing loss (conductive or nerve in any proportion) and/or shifted *downward* by any nonuniform hearing loss or by any central failure in the process of discrimination.

The distinction between hearing loss and discrimination loss is fundamental. *The shift downward (discrimination loss) is due to a failure of sense organ, nerve or brain. It cannot be offset by mere increase in loudness; but sometimes it can be offset, wholly or in part, by psychological factors such as auditory training.* The shift to the right is, by definition, a hearing loss that *can* be offset by an increase in loudness. A hearing aid can do this, provided it does not at the same time distort the speech patterns and thereby introduce a discrimination loss of its own. Fenestration, as we shall see, can cause an increase in effective loudness and shift the curve back to the left, but only within very definite limits. *Neither fenestration nor a hearing aid can significantly improve the maximum discrimination (except perhaps indirectly through psychological factors).* They certainly cannot restore missing sensory cells or nerve fibres. They may, however, move a depressed curve to the left, into the ordinary speech range, and thus give the patient the benefit of the powers of discrimination that he already possesses.

Those who have worked with deaf patients will recognize at once the two extreme types of deafness suggested above. One is the man with conduction deafness who says, "Louder, please," and understands perfectly when you shout at him. The other, nerve deaf and dropping the sibilants in his own speech, says, "Don't shout. I hear you, but I can't understand what you say."

OTHER TESTS OF HEARING LOSS.

The hearing loss for speech can be measured by many tests other than the PB lists, and practically several of them are superior for this purpose. It is laborious, time-consuming and fatiguing to the patient to give PB tests at enough different intensities to construct an entire articulation curve. Test No. 9* (spondee words), Test No. 12* (simple sentences), the 4C† records (two-digit numbers) and the Threshold of Intelligibility for Connected Discourse (TICD) are designed

*Psycho-Acoustic Laboratory. Recordings available at cost from Central Institute.

†Western Electric Co.

to measure *hearing loss for speech* accurately and rapidly, and any one of them is preferable to the PB test for this purpose.[13]

Fig. 1 summarizes graphically the relationships of all these tests to the articulation area. All of the tests were administered with the same apparatus at Central Institute for the Deaf. Much of the data has already been published, but the normal values for No. 9 and No. 12 and the PB scores corresponding to these thresholds have been verified again with a new group of 10 normal listeners, and so have the corresponding relationships between No. 9, No. 12 and the PB scores for 10 selected and experienced hard-of-hearing listeners.[3] The relationships between the tests given in Table I and illustrated in Fig. 1 are valid both for normal listeners and for hard-of-hearing listeners whose auditory discrimination, mental alertness and understanding are good enough to allow them to understand easy words and simple sentences.

In the last column of Table I is given the percentages of PB words correctly understood when the PB lists are given at the threshold levels of each of these tests. These are the ordinate values at which in Fig. 1 the horizontal lines corresponding to each test are drawn. In some cases these equivalences were determined directly, by determining a subject's threshold for connected discourse and then giving him a PB test at the same intensity. In other cases the physical value of the threshold was located on the horizontal axis of the articulation area, as in Fig. 1, and a vertical line drawn at this intensity to intersect the normal PB articulation curve. The PB scores determined in this way agreed well with those determined directly in the other type of test. The values shown in Fig. 1 and in Table I, rounded off to the nearest decibel, are those which best satisfy all of our measurements.

We can employ any of the four threshold tests (No. 9, No. 12, 4C or Connected Discourse) *to locate the position of the foot of a subject's articulation curve. His hearing loss is measured by whichever test seems most appropriate for the particular patient and the result is plotted on the corresponding scale of hearing loss.* For example, if a listener understands 50 per cent of the spondee words of Test No. 9 at 22 db

TABLE I. THRESHOLDS OF SPEECH TESTS.
(Monaural: Listening under a receiver).

Test	Number of Ears	Type of Ear	Reference	Db re‡ 0.0002 Dyne/cm²	S. D.	Accepted§ Normal Db	PB Equivalent in %
No. 9*	50	Normal	(8)	22.46	±2.63	22	8
	20	Normal	(3)	19.55†	2.72		
No. 12	20	Normal	(3)	26.65	3.89	26	23
TICD	50	Normal	(8)	23.23	3.77	23	12
PB	14	Normal	(4)	33.2	?	33	50
"Detectability"	14	Normal	(4)	15.0		13	
	?	Normal	(10)	About 10.0			
"Perceptibility"	?	Normal	(10)	About 18.0			
Intelligibility	?	Normal	(10)	About 22.0			
PB	14	Slope of normal curve, lower half	(4)		4%/db	3.85%/db	
	?	Slope of normal curve, lower half	(9)		4%/db		

*Corrected to average difficulty (cf. reference 9).
†Believed to be too low. Leads to inconsistent results in intercomparisons with other tests.
‡Based on receiver calibration in a 6 cc. coupler.
§Accepted normal, rounded to nearest decibel, used in Fig. 1.

TABLE II. INTER-RELATIONSHIPS OF SPEECH TESTS.

As Measured Directly on the Same Subjects (Monaural receiver listening unless otherwise specified).

Tests	Number of Ears	Type of Ear	Reference	Difference in Decibels	S. E.	Accepted Value, Db
No. 12—No. 9	?	Normal	(9)	"3 or 4"		
	20	Normal, field listening	(3)	4.40	±0.70	
	20	Normal	(3)	7.10*	1.00	4
	20	HOH‡	(3)	5.58	0.63	
	630	HOH	(13)	2.85	?	
TICD—No. 9	50	Normal	(8)	0.77		1
	94	HOH	(8)	About 1		
	81	HOH	(4)	2.8§		
4C—No. 9	243	HOH	(13)	About 4		4
Detectability—No. 9	237	HOH	(13)	5.05		9
	14	Normal	(4)	9.0		
PB—No. 9	152	HOH	(13)	11.25†		11
	10	Normal (Binaural, field listening)	(3)	10.86†		
Receiver—Free Field	8	Normal, No. 9	(3)	2.05*	±0.58	5
(Monaural)	20	Normal, No. 12	(3)	4.75	±0.69	5

*Inconsistent. Value for No. 9 probably too low. See Table I.
†Calculated from other measurements.
‡Hard of Hearing.
§Our recent tests show that our test for the Threshold of Intelligibility for Connected Discourse is relatively more difficult for hard-of-hearing listeners than was originally reported by Falconer and Davis.

re 0.0002 dyne/cm² (the normal value), he will understand 8 per cent of the PB words at this same intensity. If his threshold for No. 9 is at 62 db (40 db hearing loss for speech), he will also understand 8 per cent of PB words at 62 db. This point on the chart, located by plotting 40 db hearing loss for speech on the horizontal line corresponding to Test No. 9, is one point on the patient's articulation curve. His curve will be *more accurately* located in this way than by giving him a single PB list at a low intensity level, as the standard error of measurement is less. The threshold tests are also quicker to administer. Notice that the position of the curve will be the same, whichever threshold test is employed to fix its position, unless the patient has a severe discrimination loss.

It is not necessary to calibrate the apparatus in physical units in order to locate a subject's articulation curve in the Articulation Area. It is only necessary to calibrate the apparatus by finding the dial readings that correspond to the normal thresholds for the tests chosen.[9] A group of 10 young adults with no known otological defects will suffice for this purpose. A patient's hearing loss for speech is then found by subtracting the normal threshold reading from the reading obtained for the patient. This hearing loss is then plotted on the appropriate horizontal scale of hearing loss in Fig. 1. An articulation curve of normal shape is then drawn through this point.

Two points, but only two, are needed to locate a patient's articulation curve. We must know not only a hearing loss for speech but also the level of his plateau, i.e., his discrimination loss at high intensity. The discrimination loss tells how much the curve is displaced downward. *We have at present no substitute for the PB list for measuring the discrimination loss at high intensity, i.e., the downward shift of the plateau.* The exact intensity at which the minimum discrimination loss (or "PB maximum score") is measured is not critical. It is only necessary that it be *at least 35 db above the patient's threshold for Test No. 9.*

In practice we have found it convenient to administer the PB discrimination test at 110 db re 0.0002 dyne/cm² (*i.e.*, 88 db above the normal No. 9 threshold) as routine unless the patient's hearing loss for speech is more than 55 db. If his loss is 60 db or more, 120 db may be used unless it causes discomfort. Approximate but useful fitting of the curve can still be made if the level of the discrimination test is only 25 db above the No. 9 threshold.

If a patient has a discrimination loss of more than 60 per cent it becomes more and more difficult to measure his hearing loss for speech. The latter can only be measured by test material that can be understood when it is delivered loudly enough. Tests No. 12, No. 9 and 4C are usually

(but not always) well enough understood to be suitable, even in severe cases of abrupt high-tone nerve deafness.

The actual fitting of an articulation curve to the two experimental points is very conveniently and accurately done by means of a transparent lucite form or template cut to the size and shape of a normal curve drawn on a standard chart like Fig. 1. We originally prepared the prototype of Fig. 1 as a work-sheet for this purpose.

The shift of the articulation curve makes a functional but not an anatomical diagnosis. Pure loss for speech can be produced by conductive deafness, by uniform nerve deafness, and perhaps even by psychological causes. It would still be desirable to have a test that would measure conductive deafness reliably in the presence of a moderate amount of "flat" nerve deafness. Of course, a hearing aid can overcome a "flat" nerve deafness as well as conductive deafness, but the fenestration operation can overcome only the conductive element.

THE SOCIAL ADEQUACY INDEX FOR HEARING.

A patient's articulation curve gives a complete description of his ability to hear speech at all intensity levels. For example, a patient with a curve like the right-hand solid curve with a hearing loss for speech of 44 db and no discrimination loss will not recognize any words that reach him at the "faint" speech level. He will miss about 75 per cent of isolated monosyllables at the ordinary conversational level, — but he will still just barely be able to follow simple connected discourse at this level. If speech is loud, however, his discrimination loss is only about 25 per cent, and he will do very well indeed. Only unfamiliar words such as proper names will cause him any real difficulty.

The "social adequacy index" is an average of the percentage of words heard correctly at the three standard levels of speech. The index for the curve illustrated would be 33. This is the value determined as the *threshold of social adequacy* by Silverman, Thurlow, Davis and Walsh.[12]

Another articulation curve with a social adequacy index of 33 is also shown in Fig. 1. The hearing loss for speech (by No. 9) is 36 and the discrimination loss is 40 per cent. This

patient, who probably would have a mixed type of hearing loss with considerable high-tone nerve deafness, hears better than the first (nearly 50 per cent correct) at ordinary levels, but less well at the loud level. The second patient hears loud speech very little better than he hears average speech. He will not say, like the first patient, that he "hears better in noise," *i.e.*, when talkers speak loudly on account of ambient

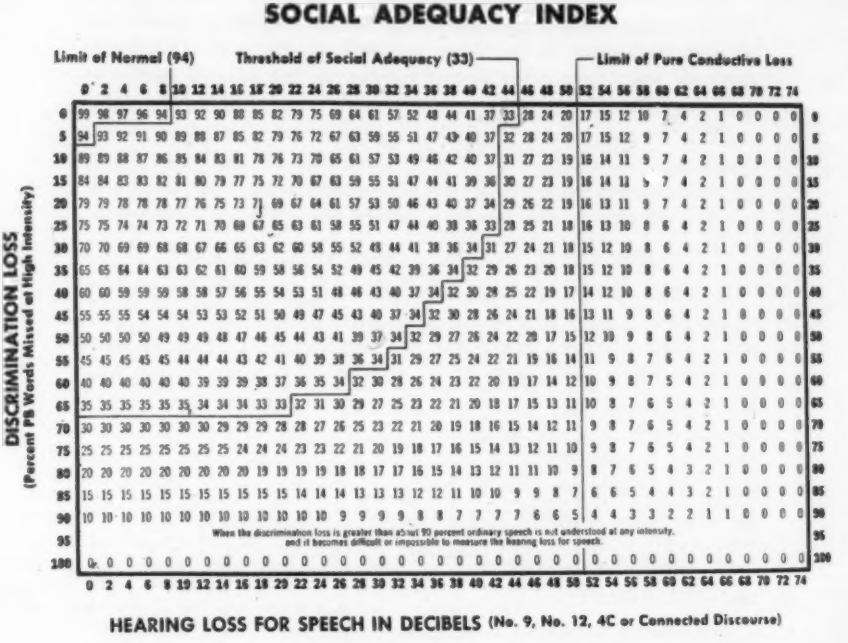

Fig. 2.

noise. In each situation the second patient "gets by" on the average, but with difficulty.

The social adequacy index corresponding to a given articulation curve may be derived graphically as described above. However, since each articulation curve is located by means of

two and only two tests (the hearing loss for speech and the discrimination loss), we have constructed a table of the social adequacy index for all combinations of these two variables (see Fig. 2). The plotting and averaging have been done in advance, and a few irregularities smoothed out. The table is very convenient to use. *With this table it is not necessary to draw any articulation curves to find the social adequacy index;* although it may be very useful to draw such a curve in order to explain to an intelligent patient the nature of his handicap and the prospective benefits and the limitations of a hearing aid or of the fenestration operation. In Fig. 2 it is obvious that the gain in social adequacy index will be greater for a given number of decibels of acoustic gain if the discrimination loss is small than if it is large. This principle is very important in predicting the benefits of the fenestration operation because the acoustic gain that can reasonably be expected is only about 35 db at most. A shift to the left of 35 db can give a tremendous improvement in social adequacy, from SAI = 7 to SAI = 70, to an ear with a 60 db hearing loss for speech and very little discrimination loss; but if the ear also has a discrimination loss of 65 per cent, the same 35 db of acoustic gain will improve the index from five to only 31. It will not even bring the ear up to the threshold of social adequacy. The fenestration operation reduces hearing loss but it does not improve discrimination.[12,13]

TABLE III. THRESHOLDS OF HEARING.*

Threshold	Db Loss for Speech†	SAI‡
1. Normal	0	98
2. Limit of normal	9§	94
3. Social difficulty begins	30	67
4. Threshold of social adequacy (middle of threshold zone)	45	33
5. Limit of compensation	60	10-15
6. Limit of compensation with a hearing aid	Still to be determined	

*From Reference 13.
†These values apply only to ears with perfect discrimination, i.e., ears that can hear correctly all PB words if they are made loud enough.
‡The Social Adequacy Index takes into account loss of discrimination as well as loss of sensitivity.
§This limit is three times the standard deviation for a group of normal listeners (see p. 765).

In Table III we have indicated the Social Adequacy Index (SAI) corresponding to a hearing loss of 9 db, the limit of

normal hearing. This SAI is 94. We have also indicated the threshold of social adequacy at SAI = 33, and (in Fig. 2) the probable limit of pure conductive deafness at a hearing loss for speech of 50 db. To these can now be added two other thresholds identified by Silverman, Walsh, Thurlow and Davis,[12] namely, the "beginning of social difficulty" at SAI = 67 and the "limit of compensation" at SAI = 10 or 15.

The social adequacy index can, of course, be derived either for a single ear from measurements made under a receiver (with masking of the opposite ear if necessary) or for the patient as a whole by binaural measurements in a free field. (For free field measurements it is necessary to use the free field threshold determined for the apparatus and for the particular test employed to find the hearing loss. The values of the SAI for a given hearing loss are the same, however the hearing loss is measured.) The index is therefore immediately applicable to the problems of *1.* fitting hearing aids, *2.* evaluating the overall benefit to a patient of the fenestration operation, and *3.* assessment of a patient's hearing for medicolegal purposes. These are all free-field problems. It can also serve for evaluation of the improvement of a single ear by the fenestration operation. This is a one-ear, under-the-receiver problem.

The apparatus required for measuring the social adequacy index is simpler for monaural than for binaural testing. In the latter case a good loudspeaker, a more powerful amplifier and a sound-treated room are needed instead of merely a good receiver. For either monaural or binaural tests a turntable, a high-fidelity pick-up and amplifier, a meter and an attenuator are required, such as we employ at Central Institute for the Deaf and described by Walsh and Silverman.[14]

SUMMARY.

The Social Adequacy* Index is a single number that indicates how well a person hears speech under average everyday conditions. It is suitable for measuring the improvement of hearing by a hearing aid, by the fenestration operation or

*"Social adequacy" throughout this paper refers always to **Social Adequacy of Hearing.**

any other therapeutic procedures, and also for medicolegal purposes.

The Social Adequacy Index is based on two different kinds of speech tests. No inferences from pure-tone tests are required. One kind of speech test measures the *hearing loss for speech* in decibels. This is a measure of auditory acuity. Several recorded tests, both words and sentences, including Tests Nos. 9 and 12 (Psycho-Acoustic Laboratory) and No. 4C (Western Electric), are available and appropriate for this purpose. The other kind of test measures the *discrimination loss*, *i.e.*, the inability to recognize difficult words even when they are spoken loudly. Discrimination loss may depend on high-tone hearing loss and/or on psychological factors. Our only quantitative tests for it at present are the phonetically balanced (PB) word lists given at high intensity levels.

From the results of one test of each kind the Social Adequacy Index may be found graphically from a chart of the Articulation Area (see Fig. 1), or, more simply, from a table (see Fig. 2). The chart of the articulation area shows how the Social Adequacy Index (SAI) is derived from the relation of the patient's articulation curve to the faint, the average and the loud speech levels that are encountered in everyday life.

The Threshold of Social Adequacy, where a person still "gets by," but with considerable difficulty, is at SAI = 33, meaning that he understands an average of one-third of the (PB) monosyllables that he hears under average conditions. Difficulty in social situations begins, however, when the index falls from normal (94 to 100) to 67, *i.e.*, when two-thirds of the PB monosyllables are still understood. Hearing becomes socially useless at an SAI of 10 or 15 when simple sentences spoken in a very loud voice are no longer understood.

A hearing aid and perhaps the fenestration operation may restore to useful levels an SAI that is reduced because of conductive deafness. Neither procedure, however, can restore an SAI that is reduced simply by a discrimination loss. Only educational procedures such as auditory training can help in this situation. A knowledge of the relative amounts of hearing loss in decibels and of discrimination loss allows a much

more accurate prognosis of the benefits of either a hearing aid or a fenestration operation.

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NONSURGICAL MANAGEMENT OF ETHMOIDITIS.*†

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The present generation of rhinologists has a rich heritage in the accumulation of successes and failures of their predecessors. The early interest in anatomy simplified the study and appreciation of this important subject. Unfortunately, along with this interest was an apparent disregard of two other equally important subjects, nasal physiology and histopathology, and this accounts for the adoption of many unfortunate therapeutic measures and the introduction of false concepts and theorems, many of which have held to the present time.

These all might be grouped together as the folklore of rhinology. Most of the items in this collection are of indefinite origin. Some seem to be the result of sound reasoning, others give the impression of being the outcome of valid research; but most of them have no scientific basis whatever. They have come down from the past, ill-founded, ill-advised, yet are taken for granted and are passed on from writer to writer and from teacher to student with perfect equanimity. From time to time one of these accepted facts of questionable origin and authenticity comes up for investigation and fails to stand up in the light of our present-day understanding of the basic principles governing the nasal and sinus cavities. It is a sign of progress when one of the accepted doctrines of the past is disproved and discarded forthwith.

In rhinology, we constantly are being confronted by theorems, dictums and customs which have had a profound influence on our behavior in the management of nasal and sinus

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conditions. Many of these seem to invite investigation. I have collected a few typical examples of rhinologic folklore which have been with us for decades, which are constantly in the literature and yet seem to be devoid of any apparent scientific foundation. Such examples are numerous and cover a wide range, but in this paper I shall deal only with that area concerning which there seems to be the greatest fund of misinformation, the ethmoid labyrinth.

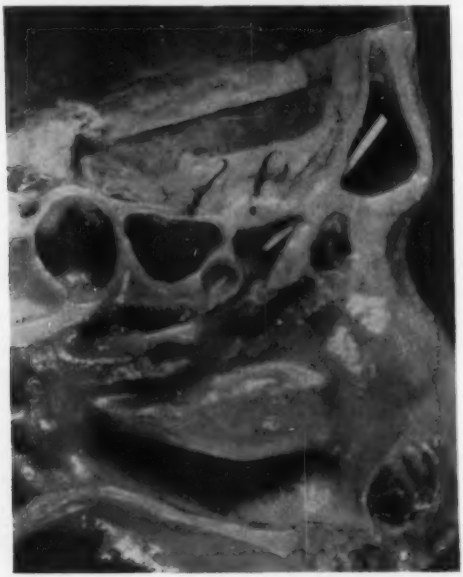

Fig. 1.

The first concerns the ethmofrontal relationship. There apparently is an idea prevalent in many quarters that removal of the ethmoid bullar cells improves frontal sinus drainage, that the approach to the frontal is often by way of these cells and that these cells are involved with all cases of frontal sinusitis.

It is true that an occasional frontal or infundibular cell encroaches on the drainage area of the frontal sinus, but only

in rare instances is a cell draining through the bullar ostium involved in this relationship. In approximately 1 per cent of cases the frontal sinus takes origin from the suprabullar groove, but no channel or duct is formed and drainage in these cases is more likely over the uncinate process into the middle meatus. Hajek[1] in his book describes one specimen in which the frontal sinus drained directly into the bulla, and the writer has in his collection a similar specimen (see Fig. 1). As far as is known, no other such cases have been reported.

The common site of drainage of the frontal sinus is into the frontal recess at a point anterior to all of the ethmoid cells and having no relation with them whatever. In the event that the drainage space is encroached upon by a cell, frontal or infundibular, obliteration of the cell is often imperative for the establishment of proper frontal drainage, but this entails no involved surgical procedure. Such a cell is usually broken down by the passage of a cannula on its way to the frontal ostium or by the use of a small rasp carefully applied. A wide removal of ethmoid cells, with or without portions of the middle turbinate, in no way improves frontal drainage and in most cases this sort of disturbance of middle meatal anatomy merely opens up other areas to be added to the infection problem.

The second item on the list concerns the relationship or lack of relationship between the anterior and posterior ethmoid cells. Rhinologists customarily lump all groups together under one heading, "the ethmoids." They treat their patients for "ethmoiditis" and schedule operations for exenteration of "the ethmoids," and the assumption is that all cells are diseased, or at least all are to be removed. The anterior and posterior groups lie adjacent to each other, yet are completely disconnected by a bony plate, the ground lamella of the middle turbinate bone (see Fig. 2). The drainage areas of the two groups are at considerable distance from one another and the likelihood of the two areas being involved in a similar infection is remote. Close sinus relationship is not uncommon, but is contingent upon the proximity of their respective drainage points; thus we expect an involvement of infundibular cells

with antral infections because of a common drainage groove, the infundibulum, and the frontal sinus may also be included in those cases in which the frontal drainage outlet is continuous with the infundibulum. The posterior cells are often suspected of being involved with the sphenoid in an inflammatory process, yet there is no anatomic justification for this assumption. The posterior ethmoids enjoy an ideal drainage system downward into protected grooves which are below and anterior to the sphenoethmoid recess, the drainage point of the sphenoid. It is true that on occasions a multisinusitis

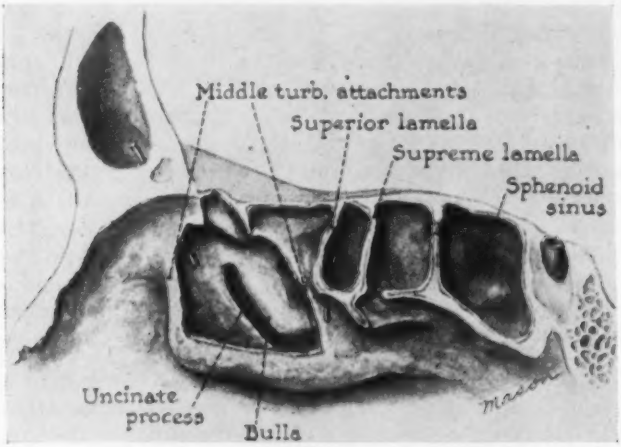

Fig. 2.

develops in which any or all cavities may be involved, and similarly in allergic conditions the lining membranes of all sinuses may reveal an edematous reaction, but in neither instance could the similarity of involvement be attributed to sinus relationship.

The habit in rhinology to regard the entire ethmoid labyrinth as a single unit of cells is unfortunate, for it leads surgeons to open up areas entirely devoid of infection.

The third item on my list concerns also the ethmoids, but deals primarily with nasal polyps. To many surgeons, the

presence of these growths in the nose is an indication for an exenteration of the ethmoids. This is based on the theory that the polyps arise from within the ethmoid cells and that all

Fig. 3.

cells must be removed for their proper elimination. This statement is false on both issues. Polyps seldom arise from the cells, and cell removal seldom brings about an eradication of the disease.

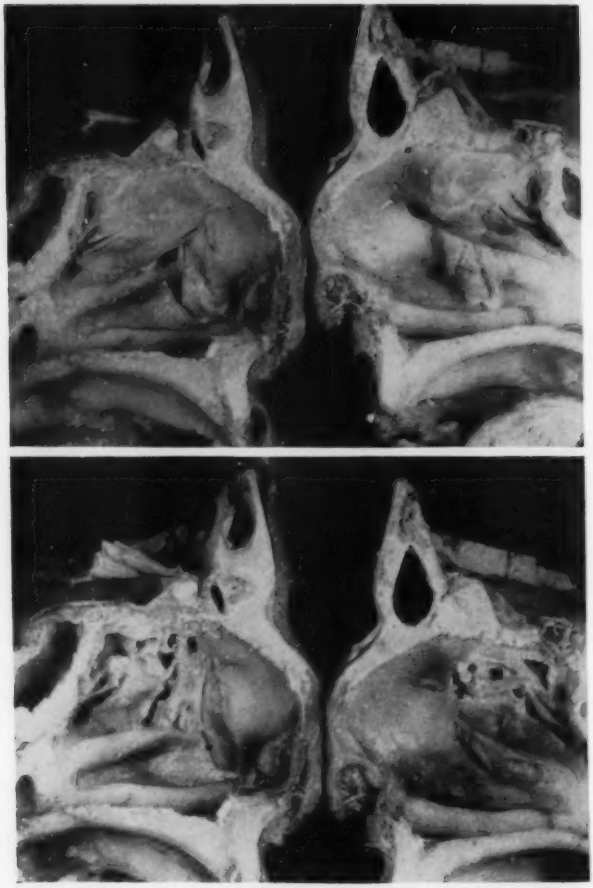

Fig. 4.

Polyps develop as a result of irritation of the nasal mucosa. The irritant may be an allergen, a pathogen or a toxic exudate from a draining sinusitis. They may arise from almost any area of the nose, but the common sites of origin are not in the ethmoid cells but from the exposed areas of the middle meatus. Polyps commonly form on the crest of the uncinate process, the margins of the maxillary ostium and the anterior surface of the outer wall of the bulla (see Fig. 1). This fact is revealed by study of large groups of specimens and is apparent to any investigator who has carefully made such a study. The writer has observed many examples of polyps in these areas, but has on no occasion seen evidence of a single polyp arising from within an ethmoid cell. It is true that the mucosa lining ethmoid cells may become edematous even to the point of completely obliterating the cell lumen, as does the lining of the antrum in cases of allergy or infection, but this swelling recedes with removal of the irritating factor, without prolapse of the tissue into the nose in the form of polyps.

Zuckerkandl[2] pointed this out years ago, and more recently his findings have been corroborated by Hajek, Hirsch,[3] Franz Wilker[4] and others.

Zuckerkandl's study of nasal polyps was made on material obtained in over 300 fresh autopsies. He stated that polyps arise from rough surfaces on the outer walls of cells such as the ethmoid bulla from the margins of sinus ostia, from grooves such as the infundibulum, from the uncinate process and from the posterior nasal sulcus, the area otherwise known as the sphenoethmoidal recess. He makes no mention of ever having observed polyps within the ethmoid cells themselves.

The final item to be considered naturally follows the last one. This deals with the premise that surgical removal offers the only possible solution to the ethmoid problem, whether the case be that of an inflammatory or noninflammatory process. The operation for removal of ethmoid cells, regardless of technique or surgical skill, has never been a highly successful procedure. This is demonstrated by the variety of approaches and techniques employed and the vast number of cases which require reoperation. Techniques are constantly being

described with the hope that they will be an improvement over the other established techniques. The various approaches in use have their advocates. Available are the intranasal, the external and the transantral approaches. The intranasal procedure of Mosher was the operation of choice for a number of years until a revision was introduced which called for the preservation of the middle turbinate. A few years ago the old Jansen-Ritter external approach was revived and temporarily, at least, obtained many followers. The enthusiasm of the 1930's for this radical operation has diminished considerably in recent years, and the intranasal method is again the favored one. Some writers advocate the transantral approach and claim for this method an opportunity for a more nearly complete obliteration of cells.

The ethmoid operation is advocated for all types of conditions involving these cells, whether it be an active inflammatory state, a so-called hyperplastic ethmoiditis, with or without polyps in the middle meatus, or a theoretical condition called catarrhal, or nonsuppurative ethmoiditis supposed to be responsible for a persistent postnasal discharge, a daily elevation of temperature, or vague pain of undetermined origin, in the face or elsewhere in the body. The operation fails of its goal in most cases and, being a destructive procedure, the usual sequela is a mutilated nose plus an exaggeration of the symptoms, with the addition of others.

Following exenteration of the ethmoids for the treatment of nasal polyps, there may be a period of freedom from the growths. This is usually not permanent, for the factors which caused them have not been eliminated and their eventual return may be predicted. In certain cases the entire middle meatus becomes filled with edematous tissue and along with this is a tendency to infection, a complication which may not have been present previous to the operation.

In justice to the surgeons who commonly resort to radical procedures in the treatment of ethmoiditis, it should be said that as a rule they have been driven to surgical intervention only after an apparent failure of the accepted conservative

measures to effect relief. It is true that the methods of the past and many in current use have been inadequate in coping with persistent ethmoiditis of one form or another, and this is due to the fact that most of them are devoid of a proper scientific foundation.

Heading the list of ineffective procedures is nasal tamponage. Tampons of mild silver protein have been used interminably on individuals suffering from all manner of nasal and sinus disease and the best that can be said for them is that they supply the physician with something to do. The principal objection to their use is that physicians who commonly use this method apply it to all cases, regardless of the diagnosis, and as a consequence are prone to develop loose diagnostic habits. As far as is known, no single case of chronic snusitis has ever been cured or tendered more than temporary amelioration by the use of argyrol tampons.

The same may be said of shrinking nose drops. In the hands of a physician for office treatment, they are of great service and there are times when their use by self-medication is definitely indicated for the temporary relief of the annoyance of a blocked nose or as an aid in the establishment of drainage in acute and subacute infections; but as a curative measure in chronic sinusitis they are of no value whatever. Included with these drops are those containing sulfonamide and antibiotics. The belief that they could be effective in the long range treatment of sinusitis when sprayed or instilled into the nose is not the result of sound reasoning, and the same may be said of penicillin aerosol and other forms of its local application.

The list of ineffective measures used in the treatment of chronic sinusitis would not be complete without mention of shortwave diathermy and Roentgen therapy. Exaggerated claims are made for these two methods from time to time, yet at best they are ancillary forms of treatment and either used as a sole therapeutic measure affords little more than symptomatic relief.

The newer viewpoint based on a better understanding of the function and other characteristics of nasal and sinus tis-

sues calls for a readjustment of our attitude regarding the management of sinus disease. Rhinologists undertaking the cure of ethmoiditis by nonsurgical measures first must abandon any preconceived ideas they may have concerning the ineffectiveness of this form of therapy when properly applied. The importance of preservation of functioning structures must be appreciated and it should be kept in mind that the membranes lining the sinuses seldom become diseased beyond repair, but possess defensive qualities with capabilities of combating disease indefinitely and returning to normal under favorable conditions. The essentials for repair are within the grasp of the rhinologist and consist in the main of the removal of factors which are the basis for the prolongation of the disease.

Important in the management of the disease is the correct diagnosis. All cases at the outset should be properly studied in order to determine the nature and exact location of the disease.

DIAGNOSIS.

Available for diagnosis are rhinoscopy, anterior and posterior, Roentgen examination, diagnostic lavage by the displacement method and transillumination. Little should be expected from the latter method, but the others, along with the history and presenting symptoms, should suffice in establishing the location of the infectious process. Important in the Roentgen study is the submentovertical projection taken with the mouth closed. This view outlines the entire ethmoid labyrinth in its long axis and brings out the ethmosphenoid relations. Views taken after opaque media has been forced into the cells by the displacement method also help.

Lavage by this method is perhaps the most valuable single diagnostic procedure available for reaching a positive diagnosis of ethmoiditis. It is best applied in cases in which the other sinuses are known to be negative, then if pus is withdrawn or blown out soon thereafter, a positive diagnosis of ethmoiditis can be made.

Important in all cases is an allergy survey. This should be carried out if possible under the personal supervision of the rhinologist, who may correlate the results with his other findings for a more complete understanding of the case. The allergic management should also be in his hands, for only in this manner may he maintain constant surveillance over the patient and retain his complete cooperation.

TREATMENT.

It is well known that the most difficult cases to treat are those which have been subjected to surgical procedures at one time or another. Landmarks have been removed, scar tissue has developed to block drainage from remaining cells and to hide away pockets of infection. In most of these cases further surgery is definitely not indicated, and the best that can be hoped for is that which may be obtained by an occasional removal of accumulated secretions, physical therapy and that which might be accomplished by minor procedures to unblock retained secretions.

On occasions areas of the operated field are sealed off by a middle turbinate, in part or in whole adherent to the lateral wall. Improvement often results when this structure is freed and pushed toward the septum. Finally, in a large group recognition and control of allergy contribute much to the patient's comfort.

In cases not previously subjected to surgical procedures, all efforts are directed to the exact area of infection. If it has been established that the anterior cells are the ones involved, it is most likely that the infection is in the bullar cells, for with an infundibular cell infection there is usually also a maxillary sinus involvement. In any event, the middle meatus must be unblocked before effective therapy may be carried out. In most cases of chronic anterior ethmoiditis or, for that matter, infection in any of the anterior group of sinuses, an impinging middle turbinate is a barrier to sinus drainage and in many of these a high deviation of the nasal septum prevents adequate infraction of the turbinate. When this defect

is corrected, displacement therapy is carried out once or twice weekly until no purulent material is withdrawn or blown out, or until no improvement is noted after a few weeks of treatment.

The cases which fail to respond to this type of management are those in which there is some overlooked or uncontrolled contributing factor, or those in which the drainage groove or ostium is occluded by edematous changes in the lining mucosa. When this has been definitely determined, the involved cells may be uncapped individually without opening up uninvolved areas.

TREATMENT OF ETHMOIDITIS WITH POLYPOID FORMATION.

This is based fundamentally on allergy control and success in the case is contingent upon elimination of irritating factors or desensitization.

Obstructing polyps are removed and this often proves to be the first step toward the successful elimination of any existing infection. Displacement therapy is carried out as outlined above and special attention is directed toward the other sinuses for a possible co-existing infection.

Cases with recurrent multiple polyps in the middle meatus have always been regarded as purely surgical cases, and most of them eventually are treated as such. As stated previously, few patients subjected to this form of treatment have little more than edematous tissue in the ethmoid cells. These tissues tend to return to normal with the elimination or desensitization treatment and the establishment of adequate drainage areas for the cells. Certainly nothing of permanence is to be gained by disturbance of the contour of the lateral nasal wall, and even though a complete termination of the disease is not always attained by nonsurgical measures the patient is more than likely free of annoying symptoms and is perhaps more comfortable than he can hope to be following the radical procedure.

SUMMARY.

Rhinologists accept without much question many theorems, concepts, dictums and customs passed on by their predecessors. Grouped together, these may be called the folklore of rhinology.

Four examples of such as applied to the ethmoid sinuses are discussed.

The first one is the theory that the ethmoid bulla commonly impairs frontal sinus drainage. This is not the case, and removal of bullar cells is seldom indicated in the treatment of disease of the frontal sinus.

Rhinologists are inclined to regard the ethmoid as a single unit of cells rather than groups of cells separated by bony plates. The two major cell groups, anterior and posterior cells, although placed adjacent to each other, are separated by the ground lamella of the middle turbinate and the likelihood of extension of infection from one group to the other is remote. This does occur in long standing, neglected cases or those of extreme virulence, and in such case extension of the inflammatory process is wide and most any or all of the sinuses may be involved.

The theory generally accepted that nasal polyps commonly arise from ethmoid cells is false. Study of anatomic specimens indicates that common points of origin are the crest of the uncinate process, the infundibulum, the margins of sinus ostia and the nasal surfaces of ethmoid cells. The mucosa lining the cells may be edematous, but there is no tendency for the tissue to prolapse into the nose in the form of polyps. The swelling of the mucosa tends to recede with the removal of the irritating factor causing it, but at no time is ethmoid exenteration indicated in treatment of nasal polyps.

The final item discussed deals with the treatment of ethmoiditis. The ineffectiveness of most of the conservative measures of the past has led to adoption of surgical procedures in the management of the disease.

This form of treatment has also been unsatisfactory and ofttimes has left the patient in worse condition than he was preceding the operation.

The proper management by nonsurgical measures calls for:

1. Preservation of functioning structures, including sinus membranes, which should be regarded as disease-resisting membranes which tend to return to normal with elimination of the factors causing the infection, and this leads to

2. Realization of the importance of correction of drainage defects such as obstructing polyps and blocking middle turbinates.

3. Elimination of other factors such as allergy, gastrointestinal, endocrine and nutritional disturbances and infections elsewhere, especially in neighboring sinuses.

4. Consideration of the patient's habits, indulgences, temperament and psychic makeup, with correction when possible.

5. Adoption of effective measures such as displacement therapy and corrective minor surgical procedures when indicated.

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NASAL AND THROAT MEDICATION: A SURVEY OF CURRENT TRENDS.*

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At no time in the history of rhinopharyngology has there been as much tendency to change as in the field of its medication. Medicating the nasal cavity has always been popular for the local treatment of most intranasal disorders, and there is today little to indicate that the current hosannas for psychosomatic medicine will prove an irrevocable substitute for nasal medication. In the treatment of nasal sinus disease the trend toward conservatism is at a peak. Actually, the promiscuous surgical practices of the first World War era and its aftermath succumbed to a dual combination — widespread knowledge of nasal physiology and the overwhelming usage of sulfonamides and antibiotics. As for the human throat, it still remains the site for the most common surgical operation in the world. Like the nasal cavity, its ready accessibility for medication makes it a favored locale for observing therapeutic activity.

Although it may be difficult for some physicians to believe in retrospect that they were ever deluded, each medical generation is familiar with various therapies that have run the gamut of overenthusiasm and disillusionment. These are the offspring of half-truths and aborted knowledge. There is, for example, growing awareness that the pattern of nasal and sinus infections changes from year to year. Such factors as seasonal variations, differing degrees of bacterial virulence, and the disconcerting presence of metamorphic strains of bacteria and viruses with novel forms of behavior should influence the rhinologist's choice of medication; for that matter, so should drug resistance, inadequate dosage, toxic reac-

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tions and the masking of symptoms. Fortunately, unrestrained enthusiasm for penicillin and the sulfonamides in the treatment of nasal and sinus infections is gradually giving way to sober judgment and judicious selection. The purpose of this paper is not only to evaluate the benefits and damages produced by the wholesale exploitation of these drugs, but to engage in some personal reflections on a number of basic features — essentially, the local use — of nasal and throat medication.

ANIMAL EXPERIMENTATION.

Animal experimentation as a basis for observing the histophysiologic effect on nasal tissues of various nasal preparations is well recognized, but not always practiced. Although it is highly advisable to determine the histophysiologic effect of drugs on normal mucous membranes of animals before advocating their widespread use in human beings, knowledge of undesirable caustic reactions sometimes comes initially via the patient's nose. If the truth be stated bluntly, this is literally paying through the nose. Years after its position in rhinology had been liquidated by clinical trial-and-error, it was learned that when mercurochrome is introduced into the nasal cavities of experimental animals, the substance will pass through the mucous membranes of the nose and sinuses, through the turbinates, through the bony walls of the frontal sinus, and even through the dura, to discolor the cortex of the brain in much less than two hours![1]

The need for determining the innocuousness of a liquid nasal medicament other than a simple clinical observation is illustrated by the wild exaggerations made earlier in the present decade in behalf of a 5 per cent solution of highly alkaline sodium sulfathiazole in the local treatment of chronic sinusitis. Rapid disillusionment, however, came quickly with the report that this preparation was both damaging to cilia and extremely caustic to the mucous membranes with which it came in contact. More recently, two cases have been described in which chemical meningitis developed in patients after a nasal sinus had been irrigated with a suspension of tyrothricin

(1:10,000).[2] The pathologic process was recreated in animal experiments — an example of closing the garage door after the automobile had been stolen.

THE ANTIBIOTICS AND SULFONAMIDES.

Because of the spectacular results obtained by the use of sulfonamides and antibiotics in controlling infections elsewhere in the body, many physicians and patients originally had high hopes that these agents would prove equally dramatic in conquering infections of the nasal cavity and the accessory sinuses. In the haste to jump on the glamorous band wagon, the fervor of great numbers of physicians outran their memory for such matters as the normal histophysiology of mucous membranes and ciliary action.

However tempting it may be to employ antibiotics and sulfonamides locally in acute and subacute nasal infections, it must be emphasized that if cilia are functioning, the medicament is carried back to the nasopharynx in a relatively short time. On the other hand, when ciliary motility is impeded, antibiotics administered locally are sometimes of minor help in acute and subacute nasal infections, especially when medication in suitable concentration is brought into contact with vulnerable micro-organisms for an adequate period of time over a wide area of affected nasal mucous membrane; nevertheless, when infective organisms are lodged in the subepithelial tissues and the overlying epithelial cells are swollen and relatively impermeable, it is difficult to see how the therapeutic medicament can effectively penetrate to the subepithelial structures. In order to be effective, therapeutic agents must gain access to the foci which they are intended to reach.

For the most part, long standing chronic sinus infections are resistant to penicillin and sulfonamides because these drugs have difficulty in adequately penetrating the thickened, fibrous mucous membranes resulting from repeated or prolonged insults. In some instances, chronic infections may be controlled temporarily, but recurrence frequently follows upon stopping medication. Sulfonamides and penicillin supplement

each other in the treatment of some infections; it may occasionally be necessary to replace one with the other when micro-organisms develop resistant strains or when the patient acquires sensitivity to the medicament. In general, it is unwise to rely upon local therapy alone in treating virulent nasal or sinus infections. The chief reliance should be placed upon systemic therapy.

Through the years, numerous germicidal solutions have been introduced into the maxillary sinus following lavage. The very multiplicity of these agents indicates the inadequacy of almost all. Not all infections of the nasal sinuses require treatment with penicillin or sulfonamide compounds. A recent communication by Burtoff[3] re-emphasizes the importance of irrigating with an isotonic solution of sodium chloride. He found that a nonbactericidal and nonbacteriostatic agent, such as isotonic solution of sodium chloride, when used locally proved just as effective in bringing about resolution of sinusitis as sulfathiazole and penicillin solutions. Nor were the results significantly different in chronic states.

In a communication published in 1943 in the *American Journal of the Medical Sciences*, I[4] warned: "Patients employing sulfonamides locally are potentially subject to all the risks entailed by oral or parenteral administration. In a therapeutic field as new as this, physicians should be on their guard as to the possibility of such complications as allergic rhinitis, toxic dermatitis, jaundice, hematuria, blood dyscrasias, hyperpyrexia, central nervous system effects, weakness and pallor, and nausea and vomiting. Unintelligent and promiscuous usage locally of sulfonamide preparations may develop in the patient a sulfaresistant state. In general, such resistance is by no means a theoretical matter." In 1948, five years later, I have had no reason to change my mind. Any medicament, no matter how valuable, may do great harm if it is employed indiscriminately.

As customarily used, the term "resistant" is applied to a micro-organism when it requires more medication to prevent its multiplication than most other strains of the same bacterial

species. Pencillin resistance proceeds slowly in man and can ordinarily be overcome by sufficient increase in dosage. In the case of streptomycin, bacteria acquire resistance at a much more rapid rate. Today it is axiomatic that inadequate dosage and indiscriminate usage of penicillin are leading factors in breeding resistant strains of bacteria. Recently there have been reports of delayed serum types of reaction to penicillin. A substantial number of sulfonamide and penicillin reactions appear to be on an allergic basis. As in the case of the sulfonamides, promiscuous use of penicillin is unwarranted in trivial infections. Since these drugs possess known limitations, it is safe to assume that new drugs will be found in due time to supplement those already utilized in contemporary rhinology.

THE OVERMEDICATED NOSE.

Experiences of the past decade have demonstrated that it is possible to curtail greatly, if not completely, two man-made nasal diseases. Recognition of the shortcomings of mineral oil as a vehicle for nasal medicaments and its subsequent omission in numerous preparations in all likelihood have diminished the incidence of lipoid pneumonia from this source. On the other hand, if one were to propose an aphorism to describe the overmedicated nose, it would run like this: overtreatment can sometimes prove just as disastrous as no treatment. It is known, for instance, that argyrosis of the mucous membranes of the nose can be due to prolonged administration of silver salts in direct contact with the nasal mucous membranes. Since argyrosis is in effect a man-made disease, its only reasonable treatment appears to be prevention. In great measure, this has been accomplished by changes in the style of drug therapy—silver salts have given way to vasoconstrictors, sulfonamides and antibiotics.

Few drugs are more widely distributed and used than nasal vasoconstrictors. The excessive use of this type of nasal medicament for a prolonged period of time causes the clinical syndrome known as vasomotor rhinitis medicamentosa.[5] The patient who overmedicates his nose usually does so to relieve

a sensation of congestion. As the practice continues, the relief afforded becomes less pronounced; finally, little or no relief is obtained. Clinically, the mucous membranes of the overmedicated nose are often indistinguishable from tissues seen during acute allergic episodes.

So far as concerns the medical literature, most complaints appear due to some of the synthetic vasoconstrictors rather than the natural ephedrine products. Generally, the synthetic products provide a greater degree of vasoconstriction and a more prolonged effect than ephedrine. Nasal sensitivity to vasoconstrictors can be checked rapidly by discontinuing the offending medicament. No rational employment of nasal medication will countenance promiscuous overindulgence. Like lipoid pneumonia and argyrosis, vasomotor rhinitis medicamentosa is man-made; its saving grace is the promptness with which cures are established.

PHYSIOLOGIC RELIEF VS. FEIGNED RELIEF.

Nasal vasoconstrictors are among the most important drugs available to rhinologists. The sympathomimetic amines afford symptomatic relief only and are not curative. Even temporary relief from the discomfiture of nasal obstruction is a boon to the individual so afflicted and a number of physiologic nasal vasoconstrictors have come to assume a legitimate place in the therapeutic armamentarium.

Excessive use leads to physiologic abuse and, like all excesses, can produce toxic reactions; however, it is difficult to challenge seriously the concept of physiologic relief which attends the elimination of nasal congestion for varying periods of time, which promotes comfort and improves nasal ventilation and which establishes the promotion of adequate drainage from infected sinuses by the opening of obstructed ostia. Physiologic relief can be accomplished with a reasonable measure of success by an intelligent choice of nasal medication. Liquid nasal medicaments which function on a rational, physiologic basis are compatible with ciliary activity, do not vary greatly in their pH (5.5 to 6.5) from that of normal nasal secretions, are isotonic, and are noninjurious and nontoxic.

In recent years various nasal vasoconstrictors in combination with various sulfonamides and antibiotics have been introduced to the medical profession and the general public. Despite the eagerness of a few rhinologists to scamper into publication with the latest brand-new "cure," in terms of clinical evaluation it is extremely difficult for sober observers to enthuse over the alleged benefits to be gained consistently by patients receiving a form of combined intranasal liquid medication. When a nasal vasoconstrictor is combined with any of the currently established sulfonamides or antibiotics, one feature is certain: physiologic relief is obtained primarily by the shrinking component. The sulfonamide or antibiotic addition serves as a therapeutic after-image — in reality, a "talking point."

This is not to imply that combined forms of nasal medication are completely without value; occasionally, one does encounter patients who swear by, rather than at, the latest vogue in nasal medication. Nor should one be skeptical as to what the future may bring. Actually, there is a crying need for new physiologic nasal vasoconstrictors — alone or in combination with newer, more effective antibiotics — for use in the effective treatment of nasal and sinus infections. Indeed, the problem of producing new sympathomimetic amines is of great importance and deserves more attention in the pharmaceutical world.

Nowhere in the field of nasal medication is there a more convincing demonstration of the absence of physiologic principles than is found in certain nasal inhalers. The drug counters are crowded with nasal inhalers which do not serve any recognizable purpose other than lighten the public purse. A common belief is that preparations containing significant amounts of menthol possess the property of opening the nasal passages. As long ago as 1885 it was demonstrated that menthol is a topical anesthetic to the nasal mucous membranes; hence, menthol used alone gives the subjective impression of reduction in nasal obstruction. Inhaled air feels cool and the subject associates cool air with increased air flow and obtains the false impression of reduced nasal congestion. In reality,

menthol causes a narrowing of the nasal passages and increased the resistance to the stream of air passing through the nasal cavity. The hard facts of the matter are that nasal preparations leaning heavily on menthol are consistently guilty of providing feigned relief.

ANTIHISTAMINIC DRUGS.

Sufficient time has now elapsed to whittle down the clinically available antihistaminic agents from a "wonder-drug" status to its true size. Just as sulfonamides simplified the problem of acute otitis media, so have the antihistaminic drugs provided palliation in the treatment of allergic rhinitis. There is, however, one notable difference — sulfonamides and antibiotics mostly cure; antihistaminics relieve. For the relief of allergic rhinitis, the antihistaminic drugs easily equal, and in numerous instances surpass, the older therapeutic trial-horses.

Allergists maintain strongly that the specific methods of managing allergy (*i.e.*, elimination and desensitization) should not be abandoned because at the present time these are the sole hopes for achieving lasting benefits. Be that as it may, it is interesting to observe that in their published communications various allergists have made more extravagant claims as to the amount of relief obtained by antihistaminic drugs than by desensitization methods. While it is unsound to administer any drug to allergic patients on a routine basis rather than when the particular need arises, particularly when the drug is capable of producing a number of undesirable side-effects, it is reasonable to suppose that the antihistaminic era is in for a period of abuse. No one can prognosticate with certainty what will happen to certain phases of the practice of allergy when improved antihistaminic drugs with a spreading horizon of effectiveness are introduced into clinical practice. Astute allergists, like astute rhinologists, realize that they are living in a changing world.

VACCINES.

The common cold is a problem that has challenged physicians for generations and frustrated them shamefully. A major reason for failure appears to be indifference and neglect at coming to grips with the problem. Although methods for the prevention of colds have been directed along various lines, it can be stated unequivocally that none of the procedures is effective. Attempts have been made to reduce the number of colds in susceptible persons by means of vaccines containing a wide variety of micro-organisms commonly found in the respiratory tract. Controlled experiments have demonstrated that the parenteral administration of cold vaccines do not prevent colds. While small numbers of patients sometimes enthuse about the benefit obtained from a course of vaccine injections, this state of affairs is most likely produced either by the seasonal well-being of the patient or by the psychological effect induced by the hypodermic needle.

As for cold vaccines given orally, the best summary appeared four years ago in an editorial published in the *Journal of the American Medical Association*,[6] as follows: "Recent communications to the offices of the American Medical Association indicate that the prescription and sale of cold vaccines is again taking place on a large scale. This, in the face of the recognized lack of scientific evidence of the value of these preparations, is indication of irresponsibility on the part of some manufacturers of pharmaceuticals. The scientific evidence against the value of oral cold vaccines is overwhelming; consequently, individual physicians and firms who deal in pharmaceuticals and who lend themselves to the wholesale uncontrolled distribution of such preparations are perpetrating an unwarranted commercial assault on the public pocketbook."

THROAT MEDICATION.

Although at any given time more physicians peer into the throat than into the nasal cavity, it has been surprisingly free from that type of intensive physiologic investigation which,

with some measure of success, has contributed to the improvement of nasal medication. As in the case of nasal medication, a judicious union between physiology and pharmacology, tempered by comprehensive bacteriologic studies and cemented by sane clinical judgment, can provide a firm foundation for rational throat medication; moreover, the intelligent usage of sulfonamides and antibiotics by physicians demands fresh knowledge of bacteriology; modern therapeutics has wedded the science of bacteriology to applied clinical practice. The more one examines the therapy of the abnormal throat, the more firmly does this thought impress itself.

For purposes of this discussion, the term "throat" is made to apply strictly to the mesopharynx — the region bounded posteriorly by the posterior pharyngeal wall; anteriorly by the base of the tongue and by the open space directed toward the mouth; laterally by the lateral pharyngeal walls; superiorly by the nasopharynx; and inferiorly by the larynx and upper pharyngeal end of the esophagus. Within the mesopharynx lie masses of lymphoid tissue, more or less symmetrically arranged to constitute Waldeyer's ring, which through their tendency to disease produce various acute and chronic inflammations and infections (tonsillitis, pharyngitis, "sore throat," Vincent's angina and lymphoid hyperplasia, to mention an important few). The manner of their treatment is twofold: local and systemic. A survey of current trends in throat medication for nonsurgical conditions points to the importance of both approaches.

RATIONALE OF LOCAL THROAT MEDICATION.

In current years considerable doubt has been cast on the value of gargles in acute tonsillitis and acute pharyngitis. Although popular with the laity and with some manufacturers of so-called throat antiseptics, the use of a gargle has no real therapeutic foundation since, as Richards[7] points out, the very process of gargling necessitates an approximation of the posterior part of the tongue to the soft palate in order to prevent fluids from running into the larynx. Experiments with methylene blue, designed to determine how efficient gar-

gling may be, demonstrate that the dye cannot be seen beyond the third molar tooth and cannot reach the tonsillar areas. Likewise, painting the throat with antiseptic solutions during acute infections is known to have only psychic value.

Medicated lozenges have struck a minor note of popularity recently in the local treatment of diseases of the throat. The efficacy of drugs administered orally, as pastilles, lozenges or pills for local treatment of tonsillitis or pharyngitis, depends largely on the direction of salivary flow, which apparently varies in individuals and with posture. MacGregor and Long[8] conducted experiments with pastilles to estimate the amount of penicillin likely to reach affected areas at the back of the mouth. Of 50 subjects studied, some had had their tonsils removed, and the rest had tonsils ranging in all degrees of size. The pastilles used were of the same base, shape and size as penicillin pastilles but contained 1 per cent methylene blue instead ot 500 units of penicillin. The pastille, for purposes of study, was placed in the mouth in the buccal fold between the cheek and teeth and allowed to dissolve without agitation. The degree of staining of the anterior pillars of the fauces, uvula and tonsils was considerable at the end of five minutes, and much more at 15 minutes, although the tonsillar area was not as heavily stained as the other two sites. The pharynx, when reached, was only slightly stained.

These experiments appear to indicate that dyes incorporated in a gelatin base reach the tonsillar area to an extent sufficient to suggest that drugs administered in a similar base should reach the tonsils in most cases. Contrariwise, lack of staining of the pharynx in a large proportion of subjects suggests that any attempt to treat pharyngitis with drugs contained in pastilles or lozenges is frequently doomed to failure. Flow of saliva at the back of the mouth and the swallowing mechanism appear to be such that saliva does not bathe this region as effectively as the oral cavity.

Arnett[9] believes that drugs incorporated in paraffin are slowly released into the saliva when chewed. During the process of chewing and swallowing, they come into contact with

the gingiva, the pharynx and the esophagus. In order to determine the extent and degree of mucosal staining produced by the incorporation of methylene blue and gentian violet, observations were made on a small group of subjects. When the patient remained in an upright position, the dyes stained the tongue, gingival margins and buccal mucosa, but usually did not reach the posterior pharyngeal tissues and the tonsils; however, when the patient chewed while in a recumbent position, with head lower than shoulders, the tonsils and posterior pharyngeal tissues usually became stained.

While not as yet established with any degree of finality, it does seem that some of the newer techniques of introducing throat medication possess, at the very least, the ability to convey medicaments closer to the sites which are to be treated than the older process of gargling. A number of investigators have found that pharyngeal infections respond well to insufflation of sulfonamides; nevertheless, in most cases of acute, uncomplicated pharyngitis, excellent response is obtained by the intelligent use of time-tried methods of treatment; namely, rest in bed, irrigation of the throat with hot, isotonic solution of sodium chloride by means of syringes or modifications of a douching apparatus. Carefully controlled treatment with sulfonamide compounds or penicillin should be undertaken when other means of therapy appear to be inadequate.

Although it is tempting to make universal use locally of penicillin and the sulfonamides, it is important to remember that effective concentrations of these drugs cannot be maintained for long on the surface of the tonsils or pharynx because of the continual washing effect of saliva. In superficial infections of the throat, penicillin may be effective; however, its value diminishes if the micro-organisms have penetrated deeply into the tissues. In acute streptococcal tonsillitis, for example, all of the offending organisms are not found on the surface of the tonsils — actually, many of them lie deep in the tonsillar tissues. Under such circumstances the value of lozenges, troches and pastilles is dubious. While penicillin is an outstanding therapy for Vincent's fusospirochetal infection of the throat and mouth, in general it is

dangerous to rely upon local medication alone in the treatment of severe infections of the throat. The chief reliance should be placed on chemotherapy and antibiotics in adequate dosage over an adequate period of time.

So widespread is the erroneous belief of the American public and a fraction of its medical population that the use of sodium bicarbonate and other alkalis is a preventive in the treatment of the common cold that it may really be called a part of our folklore. It has been asserted by Fantus that a hydrogen ion concentration near that which is normal for the mucous membrane is of even greater importance for applications to the mucous membranes than is isotonicity. Several years ago, I[10] reported the normal physiologic values for the pH of the mucous membrane of the throat in a large group of men and women with clinically normal throats to be from 4.9 to 8.0. The vast majority fell within the acid range.

From these studies it became apparent that the normal human throat may be either on the acid side most of the time or slightly alkaline in limited instances. The claims advanced by some pharmaceutical manufacturers that their particular varieties of local throat medicaments "neutralize excess acidity in the mouth and throat" can, therefore, be regarded as an additional contribution to American folklore on the alleged values of "alkalinization" in throat ailments.

A CONCLUDING THOUGHT ON CURRENT TRENDS.

As in the case of nasal medication, inadequate dosage and indiscriminate usage of antibiotics and sulfonamide compounds in the treatment of infectious diseases of the throat can breed resistant bacterial strains. Their promiscuous use is unwarranted in trivial infections. It can be stated unequivocally that these drugs enjoy a prominent position in the ranks of throat medication. Their uses, however, are limited by the development of allergic reactions, by the sensitiveness of patients to these agents, by the acquisition of "sulfonamide-resistance" and "penicillin-resistance" states, and by their ability to mask symptoms. Their abuses, on the other hand,

are encouraged by promiscuity and by lack of intelligence in prescribing for patients with throat ailments.

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INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY, LONDON, 1949.

The British Association of Otolaryngologists is organizing the Fourth International Congress of Otolaryngology, to be held in London from July 17 to July 23, 1949. There will be further meetings, for those who wish to go, at Oxford, Cambridge and Edinburgh on July 25 and 26. It is hoped that a full academic program will be arranged, and also various social functions.

The secretaries of the National Otolaryngological Societies have been circularized and asked to send a list of their members for individual notification. Should any association not receive this letter, they should communicate with the General Secretary, F. C. W. Capps, F.R.C.S., 45, Lincoln's Inn Fields, London, W.C. 2.

THE RATIONAL APPLICATION OF BARBITURATES AS PREANESTHETIC MEDICATION.*†

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The barbiturates have proved to be one of the most useful groups of depressant drugs developed by the chemists and applicable to clinical administration. Their immediate availability, their ease of administration, and their other desirable attributes have led to routine and many times rather thoughtless application in situations and in amounts which were not in the best interests of the patient or his surgeon. Any drug or group of drugs which has widespread clinical advantages is subject to overextension of its safe and useful area of application. It is the object of this discussion to approach the problem of the administration of the barbiturates for preanesthetic medication from the standpoint of the pharmacologic actions of the drugs and thereby establish a rational and satisfactory method for their clinical application.

Otolaryngologists have used barbiturates as a part of anesthesia for their operative procedures for many years with generally good results. For the most part the drugs have been used for sedation of patients on an empirical basis established by methods of trial and error. On this basis much good has been achieved, but it can be expected that much more good can be achieved if there is an understanding of the properties of the barbiturate group and the limitations of its applicability. It is along these lines that this discussion will proceed, with ultimate elaboration upon factors important to the practical application of these drugs.

Since this discussion has to do with the use of barbiturates for preanesthetic medication, there will be no discussion of

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the use of the ultra short-acting barbiturates, such as pentothal and evipal for intravenous administration. The drugs that are most often used for premedication are the short-acting group of barbiturates, such as nembutal and seconal. This short-acting group is considered because in preanesthetic medication prompt onset of action, short duration of action and quick recovery are desirable features.

In order to obtain a better understanding of the factors influencing the proper use of the barbiturates, it is well to digress for a moment to consider certain fundamental factors governing the rational application of all preanesthetic medication. It can justifiably be proposed that the administration of drugs for sedation prior to the production of complete anesthesia by any technique (inhalation, intravenous, local, etc.) is actually the first and most important step in the total anesthetic procedure. Good anesthesia is contingent upon proper preanesthetic medication. The safety and stability of the anesthesia are directly influenced by the premedicating drugs. They are of value only if used with due regard for their influence on the anesthetic agent and technique selected. The choice of the premedicating drug is unrelated to the surgical procedure and the premedicating drug affects the surgery only insofar as it facilitates or hinders the anesthesia for that surgical procedure. The drugs used possess definite pharmacologic actions which serve useful functions, and the drugs must be administered in reference to these functions and their effect upon the patient, and the anesthetic to be used. The routine use of combinations and dosages is a practice which is condemned in favor of individualized selection of premedicating agents on the basis of the patient's physical state and the anesthetic agent and technique to be used. Haphazard, routine and thoughtless ordering of depressant drugs prior to anesthesia by individuals unfamiliar with their actions and their relation to the anesthetic agent and technique will be reflected in unsafe and unsatisfactory anesthesia.

Premedication has four main functions: *1.* psychic depression or hypnosis, *2.* heightening of the pain threshold, *3.* re-

duction of reflex irritability and metabolism, and 4. counteraction of undesirable side action of the anesthetic agent.

Psychic Depression or Hypnosis: Contemplation of surgery and anesthesia by the patient about to be introduced to the strange sights, sounds and aromas of the operating room often provokes apprehension and actual fear. Exposure of the improperly premedicated patient to the preparations for anesthesia and surgery not infrequently precipitates undesirable psychosomatic responses which affect the anesthesia adversely and actually endanger the life of the patient. Unpleasant memories retained by the patient may influence later decisions about surgery that he may have to make. Apprehension, fear, undesirable psychosomatic responses and unpleasant memories can be minimized or entirely eliminated by the proper and judicious use of premedication. Many correctly, but only moderately premedicated patients can be brought to the operating room, anesthetized, operated upon and returned to their rooms with slight or no recollection of these events. These patients may have conversed rationally prior to induction of inhalation anesthesia or during local anesthesia and still be totally amnesic. When it is suitable in relation to the anesthetic agent and technique to be employed, amnesia can be guaranteed by using larger doses of the premedicating agents. Patients who have been spared the unpleasantness of raw exposure to anesthesia and surgery are unanimous in their approval of adequate premedication, and they are infinitely better candidates for anesthesia and surgery than are unprepared patients. A cooperative and calm individual is more amenable to regional block, topical and infiltration techniques and more suitably prepared for other techniques producing complete narcosis. The forcing of anesthetic agents and techniques on the uncooperative patient frequently results in poor anesthesia and inadequate operating conditions and may terminate tragically. Induction excitement, with necessary accompanying forcible restraint, is an infrequent occurrence in the correctly prepared patient.

Heightening of the Pain Threshold: The heightening of the pain threshold is a function of preanesthetic medication par-

ticularly suited to infiltration, topical and regional techniques. It is not always advisable or feasible to obtain absolute analgesia with these techniques, and under such circumstances the comfort and safety of the patient and the convenience to the surgeon are greatly enhanced by an appreciable degree of analgesia provided by the premedicating agents. Recent laboratory investigation by Gross into the analgesic properties of morphine and similar drugs seems to indicate that actual elevation of the pain threshold is diminished or completely prevented by the concurrent administration of atropine and particularly scopolamine; however, observation of the effects secured from clinical application of the combination of morphine and scopolamine leads one to believe that if the pain threshold is not elevated, at least the response to sensations of this order is altered favorably. Patients who are prone to misinterpret touch, pressure or traction as pain are benefited by the administration of analgesic drugs. When these drugs are complemented by scopolamine the alleviation of acute distress is enhanced.

Reduction of Reflex Irritability and Metabolism: Reduction of reflex irritability is a function of preanesthetic medication aimed primarily at increasing the safety of the patient. The activity of the brain and the neuromuscular mechanisms is lessened, causing mental and physical lassitude. Actual reduction of total metabolic activity may also be accomplished. Depression of these functions reduces the total amount of agent needed to effect and maintain anesthesia. This means a more rapid induction and a more rapid recovery. It minimizes the work of those organs of the body assigned to the destruction and elimination of the anesthetic agent. Depression of these functions also reduces the concentration of the agent required to effect and maintain anesthesia. This means that the incidence of toxic reactions is reduced, because the reactions are in direct proportion to the concentration of the agent.

Counteraction of Undesirable Side Action of the Anesthetic Agent: The remaining principal function of the premedicating drugs is the counteraction of certain undesirable side

actions of the anesthetic drugs. No anesthetic drug is perfect, and it is advisable and possible to overcome some of the imperfections by the judicious use of premedication. Examples of this protection are the minimizing by scopolamine or atropine of mucus production caused by inhalation anesthesia, and the reduction of the incidence of laryngospasm associated with intravenous barbiturate anesthesia. Another example is the elimination or minimizing by barbiturates of convulsive reactions to cocaine or similar drugs.

It is well to consider these principal functions of premedication in the actual application of the drugs used for that purpose. This will simplify the process and result in more rational and satisfactory preanesthetic sedation.

After the preceding exposition of the fundamental factors influencing the use of premedicating drugs it is now possible to define more accurately the rôle of the barbiturates in that area. The properties for which the barbiturates are used are hypnosis and protection against the convulsive manifestations of a reaction to cocaine and similar drugs. It can be seen that the barbiturates are, therefore, extremely useful in otolaryngologic practice. It is well to remember that the barbiturates are poor agents for analgesia and should not be used for that purpose. They should be used with caution in patients with pain, because the barbiturates repress the inhibitions and often provoke disorientation.

Route and Time of Administration: The short-acting barbiturates are usually given by mouth, although preparations are available which can be given parenterally. There is a growing tendency to use nembutal intravenously for premedication and sedation. The use of the barbiturate intravenously avoids the unreliable effect associated with the inconstant absorption of the drug from the stomach and intestine. Intravenous administration permits the development of more exact degrees of depression. The maximal effect from nembutal given intravenously is obtained in approximately three minutes. The maximal effect is obtained in approximately 20 minutes when the drug is given by mouth. It is important to

remember that the barbiturates are absorbed best from the alkaline medium of the small intestine. This fact makes it advisable to give the barbiturate a half hour before the administration of morphine when the two drugs are used together. The morphine relaxes the stomach and increases the tone of the pylorus, thereby delaying the passage of the capsule of barbiturates from stomach to small intestine. The effect of the short-acting barbiturates lasts approximately four hours.

Reactions and Their Treatment: Unfavorable reactions to the barbiturates are due most often to overdosage, causing respiratory depression, obstruction and asphyxia. They often cause circulatory depression. The respiratory depression is characterized by shallow respirations at a normal or slightly increased rate, in contrast with the slow, deep breathing of morphine depression. In the treatment of barbiturate depression the airway must be established, the patient must be oxygenated and the circulatory depression relieved. The analeptic drugs have little or no value unless the depression is minimal, because the analeptics effect only a brief stimulation and the patient lapses again into the depression.

Another unfavorable action of the barbiturates is their tendency to increase the sensitivity of the laryngeal reflex and, therefore, to predispose to laryngospasm. For this reason it is not advisable to use them as premedication for ether.

Occasionally some individuals manifest allergic reactions to the barbiturates. These are usually characterized by cutaneous lesions. Use of barbiturates should be avoided in patients giving a history of such a reaction.

A few people become disoriented under the influence of the barbiturates, particularly patients over 70 years of age. The incidence of such reactions is low, however, and close scrutiny of a history of this type of reaction usually reveals that the barbiturates were used in the presence of pain, in excessive doses or in some other improper fashion.

Doses of Drugs: The dose of the premedicating drug is influenced, first, by the anesthetic agent and technique. Of the inhalation group, nitrous oxide and ethylene require large

doses because of their relative impotence. Ether requires only moderate doses because it is potent and reflexly stimulates respiration. With cyclopropane, only mild doses are tolerated because, although it is a potent agent, it does not reflexly stimulate respiration and apnea frequently occurs with moderate or large doses of morphine. Divinyl ether requires moderate doses. Chloroform requires mild doses. Ethyl chloride requires moderate doses.

Patients under spinal anesthesia need moderate to large doses of premedicating drugs to insure their comfort.

Patients receiving block, topical or infiltration anesthesia require large doses of premedicating drugs to insure their comfort and to complement the anesthesia. Somewhat smaller doses are used if the patient must sit for the surgical procedure.

The ultimate determination of dosage depends on the patient's level of reflex irritability. This level of reflex irritability parallels fairly constantly the metabolic activity of the individual. The metabolism of the individual varies with his age. The most important single factor influencing the estimation of the level of reflex irritability, and, therefore, the tolerance to depressant drugs, is the patient's age. If anyone asked to prescribe a depressant drug were limited to one piece of information about the patient, the most valuable item would be the age. From a given value at birth, the metabolic activity increases sharply to five years of age, when it recedes slightly, to rise again at puberty, after which it gradually declines until old age. This variation of metabolic activity delineates the reason for the frequent under-premedication of children and the over-premedication of the aged.

Basic levels of reflex irritability and metabolism are increased by fever, pain, emotional disturbances and specific hypermetabolic states (hyperthyroidism). Patients with such complications have a higher tolerance for depressant drugs and require larger doses than other patients.

Basic levels of reflex irritability and metabolism are decreased in certain races (Negroes, Orientals) and by debili-

tating diseases, asthenia and specific hypometabolic states (hypothyroidism). These persons tolerate depressant drugs less and require smaller doses than the average patient.

Barbiturates are used for premedication for anesthesia for otolaryngologic procedures under chiefly two circumstances. It is a useful drug in establishing an appreciable degree of basal narcosis prior to nitrous oxide anesthesia and it is essential as premedication for reasons outlined earlier for anesthesia by infiltration, regional or topical techniques in which cocaine or similar drugs are used.

There are many operative procedures in otolaryngology which do not require relaxation and for these procedures nitrous oxide has proved to be a very useful inhalation anesthetic agent. Some of the procedures that can be done safely and quite satisfactorily with nitrous oxide are mastoidectomies, fenestration operations, neck dissections, laryngectomies, plastic procedures about the head and neck, and oral surgical procedures. Nitrous oxide is a weak anesthetic agent but able to produce first plane, third stage anesthesia *without* reducing inspired oxygen concentration. It is capable of producing only very minor degrees of muscular relaxation. It is entirely nontoxic, and it is the safest anesthetic agent available when used with a normal supply of oxygen. It is especially useful when only mild to moderate muscular relaxation is required and/or a noninflammable agent is needed. It should not be used in concentrations higher than 80 per cent, and preferably is used in concentrations not exceeding 75 per cent.

In order to use nitrous oxide in this manner, it is necessary to reduce the reflex irritability and metabolism of the patient with some other drug or combination of drugs prior to, or coincident with, the production of anesthesia with nitrous oxide. This may be accomplished by three methods, depending on the anticipated length of the anesthesia. It may be done by using: *1.* an ultra short-acting barbiturate intravenously, *2.* relatively large doses of short-acting barbiturates and morphine and scopolamine, and *3.* tribromethanol with amylene hydrate (avertin) or ether per rectum.

If it is anticipated that the nitrous oxide anesthesia will be moderately long (one to three hours), it is expedient to effect

the depression of reflex irritability and metabolism by giving large doses of short-acting barbiturates orally or intravenously, morphine and scopolamine. The advantage in this method of depression of reflex irritability and metabolism for longer anesthetics over pentothal lies in the fact that the premedication wears off during the anesthesia and permits more prompt awakening of the patient, whereas if pentothal were given to maintain a long period of hypnosis, the accumulation of pentothal would cause protracted postanesthetic depression. Nembutal has been customarily used in this type of medication for nitrous oxide anesthesia, although other short-acting barbiturates, such as seconal, may be employed. One of the disadvantages of administration of these drugs perorally has been the relatively inconstant degree of depression owing to the variable absorption of the drug from the stomach and upper intestinal tract. Preparations of nembutal are available for administration intravenously, and their use has made it possible to achieve more precise degrees of depression. The older preparation, one used for years by the veterinary profession, is a solution containing 1 gr. (0.06 gm.) per cc. It contains a small amount of alcohol as a preservative. A newer preparation contains approximately 0.05 gm. per cc. and has alcohol and propylene glycol as preservatives. Either preparation is suitable for use in human beings.

Experience with the use of nembutal intravenously for premedication for nitrous oxide anesthesia made it evident that the drug produced degrees of narcosis which were comparable to those produced by pentothal. It was also evident that there was an appreciable diminution of the incidence of laryngospasm. Because of these two factors, nembutal given intravenously has been useful in producing narcosis prior to endotracheal intubation, in endoscopic procedures and during anesthesia by means of regional, topical, infiltration and spinal techniques.

As stated earlier, barbiturates are essential in the premedication of patients to be operated upon under local, topical or regional anesthesia. These drugs give an appreciable and beneficial degree of hypnosis and provide a certain amount of

protection against the convulsive portions of reaction to local anesthetic drugs. There are a few practical points in the use of barbiturates in these instances. If the patient is to be supine, slightly larger doses of the drug can be used than if the patient is to be operated upon in the sitting position. One must be careful to avoid overdose of the drug and in this respect there is considerable advantage in the administration of the drug by the intravenous route. As pointed out in that discussion, a more accurate estimation of the effect of the drug can be ascertained when it is given intravenously and overdoses will be less frequent.

It is advisable to administer the barbiturates in combination with morphine and scopolamine, and if the barbiturates are used in this combination prior to local anesthesia and they are administered orally, they should be given one-half hour before the morphine to provide for more adequate absorption. If they are given intravenously this precaution need not be observed.

It has been the experience of many otolaryngologists working with patients under local anesthesia to have that patient become restless and unmanageable during the operation, even though the anesthesia at the site of the operation is quite satisfactory and complete. This restlessness is often due to uncomfortable position, warmth, pressure, etc. It can often be alleviated so that the procedure can be completed carefully and with deliberation if the patient is given additional sedation intravenously at that time. This sedation can be accomplished by the intravenous administration of relatively small doses of morphine and scopolamine, but many times small additional doses of nembutal intravenously are suitable. It is emphasized that whatever medication is used, it should be given intravenously. The absorption of the drug given orally or subcutaneously or intramusclarly is so slow as to be ineffective for the immediate situation.

A brief summary of the factors influencing the rational use of the barbiturates has been presented. Close attention to these factors will serve to improve the benefits to be gained by patient and surgeon when barbiturates are used in premedication.

RUPTURE OF BRONCHUS DUE TO EXTERNAL CHEST TRAUMA; REPORT OF THREE CASES WITH RECOVERY.*†

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KENNETH C. JOHNSTON, M.D., Chicago, Ill.

Rupture of a bronchus is an unusual and serious complication of severe external trauma to the chest. Kinsella and Johnsrud[1] presented a very complete review of the topic in a recent publication and were able to uncover only 38 cases in the literature. The first report of a patient surviving this type of injury was made by Krinitski, in 1928. In this case, complete bronchial stenosis was noted as an incidental postmortem finding, the apparent result of chest trauma suffered 21 years previously. The second instance of recovery was reported in 1939 by Jones and Vinson,[2] who were the first to establish the diagnosis clinically in a nonfatal case. Recovered cases have since been reported with increasing frequency, testifying to the increasing awareness of the entity and also to the recent advances in the treatment of severe thoracic injuries.

The following three cases with recovery are presented to emphasize further the not too rare occurrence of traumatic bronchial rupture and to call attention to the complexities of treatment.

Case 1: T. T., a 24-year-old white female, was admitted to St. Luke's Hospital on Jan. 12, 1944, with the chief complaint of dyspnea. The patient had been injured in an automobile accident five weeks previously (Dec. 5, 1943), and suffered fractures of two ribs on the left, a dislocated hip and cerebral concussion. She was hospitalized for two and one-half weeks at another hospital, and during the first few days was unconscious and expelled bloody secretions from the mouth. No other details of the early post-accident course were obtainable. Convalescence was apparently uneventful except that when the patient was first allowed

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out of bed she noted shortness of breath on the slightest exertion. This dyspnea persisted until entry. Physical examination was essentially negative except for the chest findings. Impaired resonance and absent breath sounds were noted over all of the left lung field except in the upper anterior one-third. The blood count and urine were normal. The

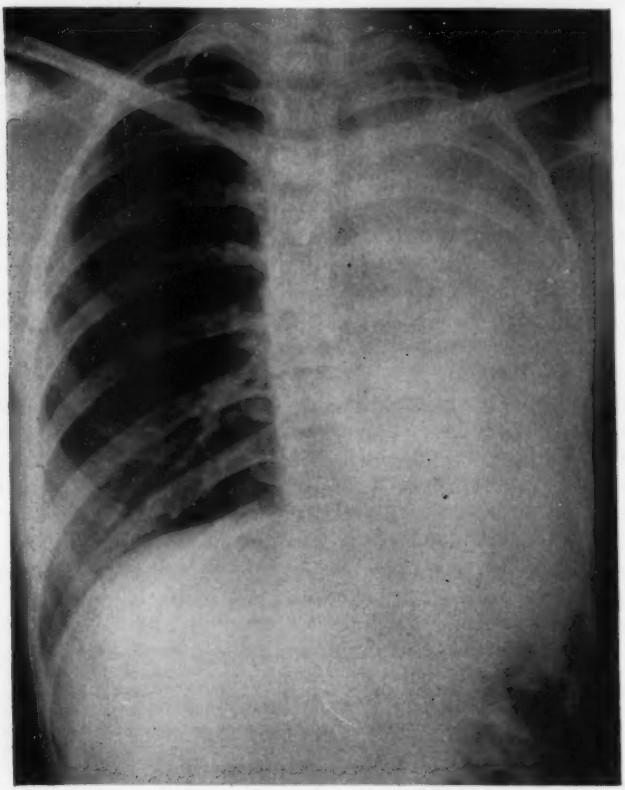

Fig. 1. Case 1. Roentgenogram of the chest showing evidence of collapse of left lung with herniation of right upper lung into left chest.

chest X-ray (see Fig. 1) showed a uniform opacity over the lower two-thirds of the left lung field, displacement of the mediastinum to the left, elevation of the left diaphragm and herniation of the right lung to the left through the anterior mediastinum. Bronchoscopy revealed the following: Immediately proximal to the upper lobe orifice, the left main bronchus was found completely obstructed by what appeared to be a

firm fibrous web. A forward grasping forceps was advanced through the web and the resultant opening gradually dilated. This permitted passage of the bronchoscope through the stenotic area to expose the left lower lobe bronchus filled with old blood. At the point of the tightest stenosis, in the region of the left upper lobe bronchus, a broken cartilaginous ring appeared through the mucosal wall. The stricture in the left main bronchus was dilated on three subsequent occasions during the next four weeks with progressively increasing difficulty. On April 5, 1944, bronchoscopy revealed the former stenosis to be completely closed and attempts to penetrate this area were unsuccessful. The patient's symptoms and findings, otherwise, remained essentially unchanged.

On April 14, 1944, an exploratory thoracotomy was done (Willard E. Van Hazel, M.D.). Both lobes of the left lung were found airless. There was evidence of trauma at the hilus, in that the mediastinal pleura could easily be dissected away from the structures in this area. The hilar vessels were intact, although somewhat difficult to identify. The left main bronchus was sectioned below the site of occlusion and the left lung removed. The procedure was tolerated well, and the patient made an uneventful recovery. Following the operation, the patient's dyspnea was improved.

Pathological examination of the resected lung revealed it to be grossly collapsed and hyperemic. Microscopically, the bronchi appeared intact and in some areas contained homogeneous material with a few scattered cells. The lung parenchyma in some sections showed alveoli in a state of simple collapse. In other areas, the alveoli were filled with a pale staining homogeneous material, containing large mononuclear phagocytes and an occasional red blood cell. There was no significant evidence of inflammatory reaction.

Comment: This patient had suffered an obvious rupture of the left main bronchus as a complication of her severe external chest trauma. The diagnosis of the bronchial injury was not established until bronchoscopic examination five weeks after the accident, at which time the occlusion of the involved bronchus was already complete. Hemorrhage, inflammatory and reparative processes were apparently involved in production of the bronchial obstruction. Collapse of the left lung and distention of the contralateral lung were natural sequelae. The absence of complicating infection in the collapsed lung, both on clinical and pathological examination, is considered significant. The exact cause of the patient's marked dyspnea could not be readily explained on the findings available.

Case 2: R. D., six-year-old white male, was admitted to Children's Memorial Hospital on Nov. 18, 1945. The patient had been struck in the left side by an automobile two months previously. He sustained a fracture of the left humerus and left femur, for which he had been hospitalized at another institution. The fractures were reduced and immobilized, and the patient discharged one week before admission to the Children's Memorial Hospital. He had remained confined to bed at home and had no respiratory symptoms other than a slight cough. Further studies had been advised because of persistent abnormalities on the chest X-rays.

Interpretation of the previous chest X-rays was as follows: Sept. 12, 1945 (approximate time of accident), "Tension pneumothorax on the left with displacement of the mediastinum to the right. No evidence of fractured ribs." Oct. 10, 1945—"Pneumothorax decreased. Mediastinal structures have returned to a position slightly to the left of the midline. There is evidence of hyperexpansion of the right lung and collapse of the left

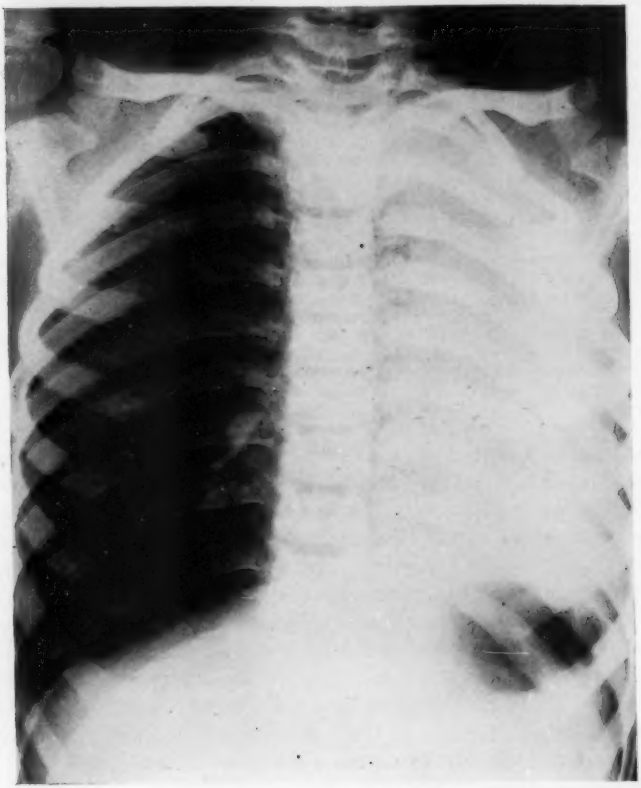

Fig. 2. Case 2. Chest Roentgenogram showing evidence of collapse of left lung.

lung." Oct. 30, 1945—"Right lung shows increased distention with herniation into left chest. Cardiac shadow is displaced markedly to the left." Physical examination revealed a poorly nourished, fairly well developed boy, not appearing ill. Temperature, pulse and respirations were normal. The left chest showed restricted expansion. There was

impaired resonance and absent breath sounds over all of the left lung except in the upper one-half anteriorly. The trachea and heart were displaced to the left. The fracture sites were apparently healing well. Blood count and urine were essentially negative. An X-ray of the

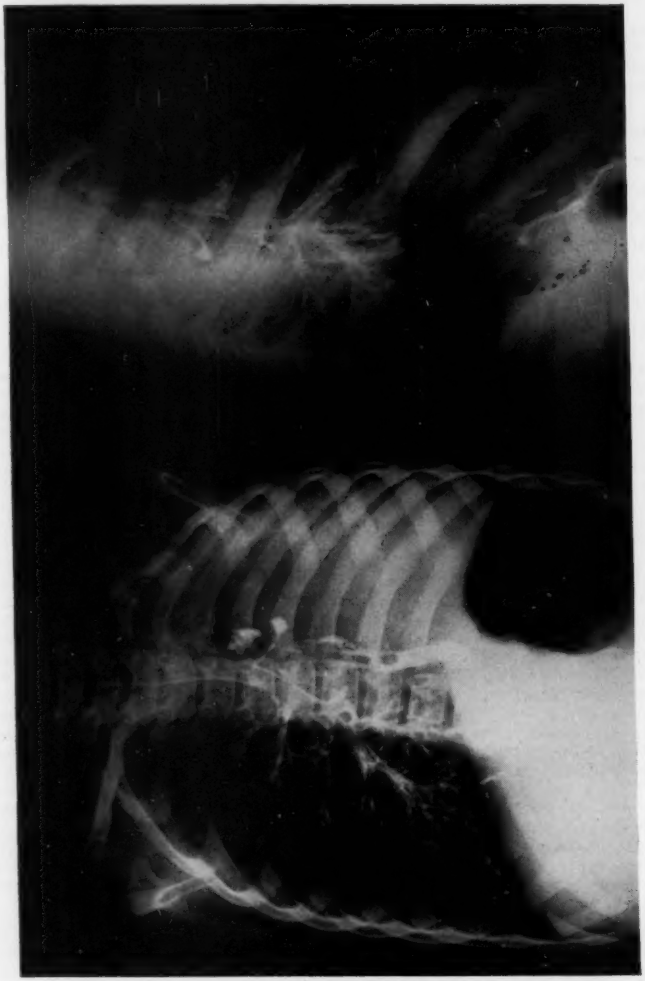

Fig. 3. Case 2. Lipiodol bronchograms demonstrating complete occlusion of left main bronchus. Posterior deviation of the occluded bronchus is noted on the lateral view.

chest (see Fig. 2), revealed a diffuse opacity of the entire left lung field, displacement of the mediastinum to the left and contraction of the left hemithorax. On bronchoscopy, the coryna was found to lie obliquely. The left main bronchus deviated posteriorly and was completely obstructed about 3 cm. below the bifurcation, by what appeared to be complete adhesions between the anterior and posterior walls. Efforts to dilate the stenotic area were unsuccessful. Bronchogram (see Fig. 3) confirmed the diagnosis of a complete occlusion of the left main bronchus. The patient remained afebrile and asymptomatic during his hospital stay and was discharged home 10 days after entry.

Comment: The findings in this case were consistent with a traumatic rupture of the left main bronchus. This was followed by complete bronchial occlusion and collapse of the left lung. These sequelae apparently developed gradually over a four to six weeks' period following the accident, as far as could be determined from the serial chest X-days. Noteworthy was the lack of any clinical evidence of pulmonary infection or disturbed pulmonary function. Physiologic studies, however, may have revealed some degree of functional impairment.

Case 3: B. J., a five-year-old white female, entered the Children's Memorial Hospital for study on Oct. 27, 1947, because of abnormal chest findings noted on a routine preschool examination. The patient was in an automobile accident three years previously and sustained a fracture of the left clavicle. She was hospitalized for one week, and for several days was said to have been cyanotic and dyspneic. A chest X-ray at that time was reported to have shown "collapse of the left lung." Several months later, another X-ray was taken and the lung was said to have been "still collapsed." The patient had no further medical attention until examined for nursery school three weeks before admission. Since the time of the accident the patient was noted to have had exertional dyspnea, an intermittent unproductive cough, and ease of fatigue. Examination revealed a listless, poorly nourished child in no evident distress. The temperature was 99.6°, the pulse 108, and respirations 24. There were findings of a mild, acute upper respiratory infection. Movements of the left chest were restricted, and dullness and suppressed breath sounds were noted over the entire left lung field, except in the upper anterior aspect. The heart was displaced to the left. The remainder of the physical examination was negative. The white blood cell count was 13,600, the hemoglobin was 14 gm. per cent, and the red blood cells numbered 4,520,000. The urine, Kahn reaction, and tuberculin were negative. X-rays of the chest (see Fig. 4A) revealed diffuse density over the lower two-thirds of the left lung field with displacement of the mediastinum to the left. On bronchoscopy, the coryna was noted to be displaced to the left, and the left main bronchus extended posteriorly more than is usually seen. At about the level of the left upper lobe orifice, an obstruction was encountered, through which the flexible tipped aspirator could not be passed. Bronchograms (see Fig. 4B) confirmed the suspicion that complete occlusion of the left main bronchus was present.

The patient was treated with penicillin (20,000 units intramuscularly every three hours) for two weeks, and the signs of the upper respiratory infection and the slight fever promptly cleared. The chest findings

remained unchanged. An exploratory thoracotomy was performed on Dec. 3, 1947 (W. J. Potts, M.D.). Both lobes of the left lung were found completely airless, each lobe measuring approximately 3 x 3 x 1 cm. in size. There were no adhesions in the left pleural cavity and the pulmonary

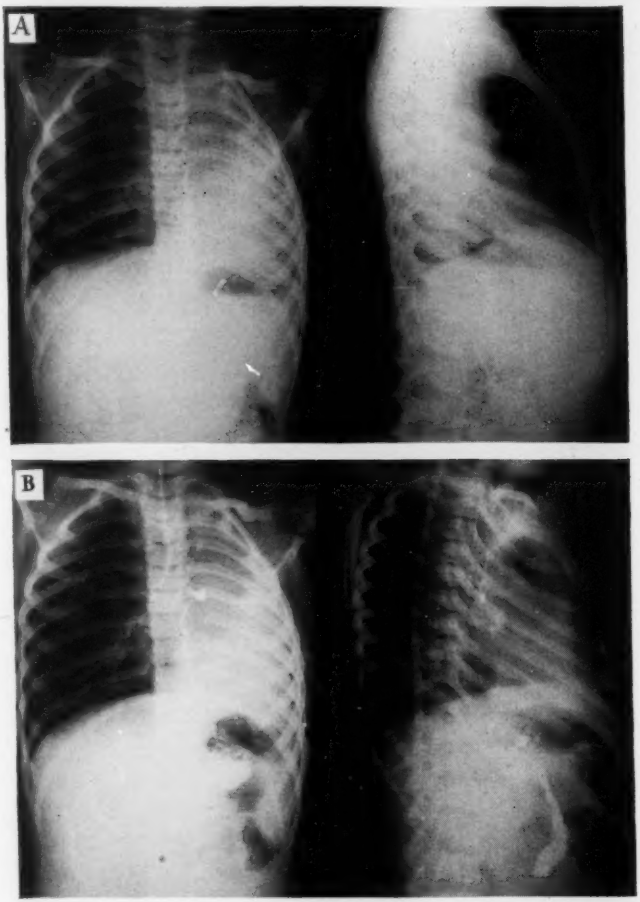

Fig. 4A. Case 3. Chest Roentgenograms showing collapse of the left lung. Herniation of the right lung to the left through the anterior mediastinum is noted on the lateral view.

Fig. 4B. Case 3. Lipiodol bronchograms demonstrating complete occlusion of the left main bronchus.

arteries and veins to the left lung were intact. The left main bronchus was found to be completely transected, with the distal and proximal portions separated. A left pneumonectomy was done and the patient's postoperative course was entirely uneventful. On a follow-up clinic visit on

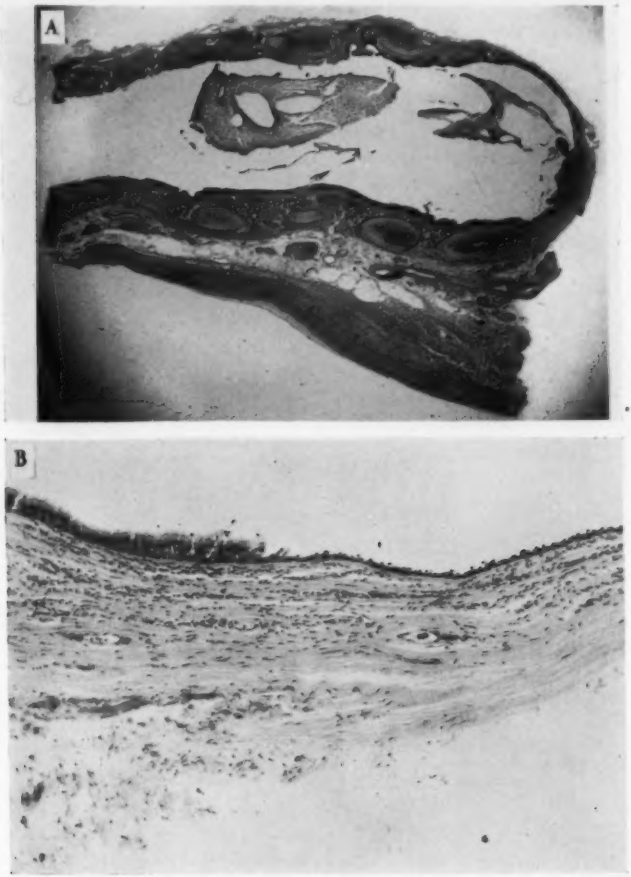

Fig. 5A. Case 3. Low power view (×13.5) of distal end of left main bronchus showing healing with complete occlusion.

Fig. 5B. Photomicrograph at reparative site demonstrating the replacement fibrosis and lining of single layer of nonciliated columnar epithelium. Normal bronchial structure is seen on left half of section.

Feb. 20, 1948, the child was reported to be feeling generally well, without cough or dyspnea.

Pathological examination of the resected lung revealed the left main bronchus to end blindly 1.2 cm. below the bifurcation. The bronchi distal to the transection were intact and filled with a coagulated homogeneous

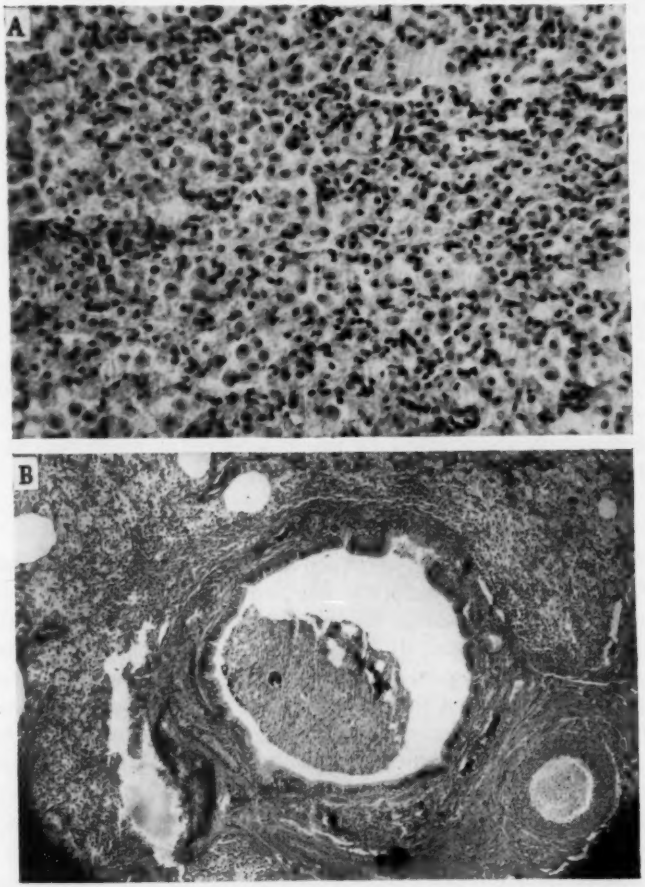

Fig. 6. Case 3. Photomicrographs of parenchyma of resected lung demonstrating: (a) simple collapse of alveoli due to nonaeration; (b) intact bronchus with coagulated mucus in lumen. No evidence of inflammatory reaction is noted in these sections.

grayish material. Both lobes of the lung were entirely collapsed. Microscopically, the terminal blind end of the left main bronchus was found to be composed of fibrous tissue without cartilaginous or glandular elements, and lined by a single layer of columnar epithelium (see Fig. 5). The homogeneous material within the lumen of the distal bronchi appeared microscopically to be coagulated mucus with a few scattered cells. The lung parenchyma (see Fig. 6) showed only completely collapsed alveoli without evidence of acute or chronic inflammatory reaction: The alveoli were easily expanded on injection of air into the lesser bronchi. The bronchi throughout appeared intact.

Comment: This case illustrates more severe bronchial injury than either of the two previous cases. Complete transection of the left main bronchus had occurred with definite separation between the proximal and the distal ends. From available data it appeared that collapse of the left lung followed immediately after the bronchial interruption, as would be expected. The clinical findings were not unlike those in the previous two patients. The process of healing by fibrosis and epithelization at the site of the bronchial fracture was clearly demonstrated. Extremely interesting was the status of the left lung on pathologic examination after three years of collapse. No evidence of infection or other pathologic change, aside from airlessness, was noted. Since, on empirical grounds, a permanently atelectatic lung was considered more or less of a hazard, pneumonectomy was done.

BASIC CONSIDERATIONS.

The exact mechanism by which external trauma to the chest produces bronchial rupture is not known. The traumata have been of severe grade in all recorded cases and automobile injuries predominate. It is noteworthy that no mention of bronchial injury is made in reported surveys of crushing chest injuries encountered in the last war. The left bronchus has been more often affected than the right and the usual site of injury is just below the coryna. There may be either a partial disruption or complete transection of the bronchus. If the individual survives the immediate injury, some degree of bronchial stenosis, usually complete, inevitably results.

The physiologic and pathologic considerations in these cases are extremely interesting. Their discussion must be largely theoretical, since no studies of the basic cardiopulmonary

mechanisms have yet been reported. The pathologic sequence of events following the bronchial injury has been fairly uniform in the recovered cases thus far described. The bronchial airway becomes permanently and completely interrupted, either instantaneously in the total transections or within a relatively short time in the partial disruptions. In either event, aeration to the affected lung ceases abruptly and rapid absorption of the trapped air with complete parenchymal collapse follows. (In the case of partial bronchial rupture reported by Tyson and Lyle,[3] total lung collapse was noted within 72 hours after the injury.) The involved lung then remains in a chronic state of simple or uncomplicated atelectasis — a rare occurrence among the variety of pathologic conditions commonly labeled "atelectasis." Pinner has stated that "uncomplicated chronic atelectasis is an extremely rare occurrence in human pathology, if indeed it ever occurs." That this condition actually does exist in these cases is demonstrable beyond doubt.

Due to the smaller space-occupying airless lung, the negative intrapleural pressure on the affected side rises markedly. Negative pressures up to —58 cm. of water were observed in the case of Tyson and Lyle.[3] There follows, then, a shift of the mediastinal structures, contraction of the hemithorax, elevation of the diaphragm and distention of the contralateral lung. These and other readjustments occasioned by the lung collapse are not unlike those following pneumonectomy and have been amply discussed by Rienhoff[4] and others.

Major circulation to the affected lung is not interrupted in the recovered cases of bronchial rupture. Passage of venous blood through the airless lung would be expected to result in some reduction in oxygen saturation of the arterial blood, but studies to determine whether this occurs are not available. Apropos are Maier's[5] remarks in Nelson's Surgery that "the blood flow through an atelectatic lung is markedly diminished. If the atelectasis is produced suddenly, a period of adjustment is necessary before this pulmonary blood flow diminishes sufficiently to prevent oxygen unsaturation of the arterial blood. Some mechanism to regulate blood flow in relation to venti-

lation apparently exists." This may be one explanation for the early transitory dyspnea noted in some of these cases under discussion.

In regard to changes in functional capacity, a number of pathophysiologic concepts, evolved from postpneumonectomy studies, would seem applicable to these patients with chronic lung collapse. Pulmonary function is influenced by the status of the opposite lung in both conditions and, in both, overinflation of the contralateral lung develops. Investigations by Cournand and his co-workers[6] indicate that, following ablation of one lung, overdistention of the remaining lung leads *per se* to varying degrees of functional impairment, depending upon its extent and upon the age of the individual. Irreversible emphysema may later develop in the distended lung to further increase dysfunction, but this point is still a controversial one. Distention and possible emphysema have been found to be more serious considerations in adults than in children. A factor of added significance in the case of collapsed lung is the increased negative intrapleural pressure on the affected side. This would tend to further increase mediastinal displacement and distention of the opposite lung. In cases reported by Clerf[7] and Tyson and Lyle,[3] reduction of the high intrapleural pressure with pneumothorax gave prompt relief of dyspnea. Pneumothorax has, of course, been used under similar circumstances in postpneumonectomy dyspnea.

The singular absence of infection in the chronically collapsed lung, in the reported cases with complete bronchial occlusion, has been a striking finding. This has been demonstrated both clinically and pathologically. Presumably it is due to the lack of significant pre-existing infection and to the barrier to subsequent bacterial invasion from above. This phenomenon has its counterpart in animal studies reported by Tannenberg and Pinner,[8] in 1942. These investigators produced complete obstruction of the main bronchus by ligation in rabbits and found that complete atelectasis ensued within a few hours. The atelectatic lungs were allowed to persist for periods up to two months without developing any complicating infection or bronchial dilatation. It would appear from these

observations in animals and humans that an atelectatic lung, when uncomplicated by infection, pre-existing disease or circulatory impairment, can exist for prolonged periods without undergoing significant pathologic change. Theoretically, if the bronchial airway could in some way be adequately restored in these patients, one might reasonably expect the collapsed lung to resume full function.

The few reported cases in which bronchial injury resulted in an incomplete or partial stenosis must be considered in a separate category. The pathologic sequelae in this group would be the same as in cases of partial bronchial obstruction from any one of a number of other causes, and are too well known to warrant any further comment.

DIAGNOSIS.

Diagnosis of bronchial rupture during the period immediately following the major trauma would appear to be exceedingly difficult and has rarely been made clinically. Other concomitant injuries to the thoracic cage and viscera usually obscure and complicate the picture. No single or composite clinical findings are pathognomonic of a bronchial injury at this stage. The use of bronchoscopy solely to prove the presence of a bronchial rupture, although recommended by Kinsella and Johnsrud,[1] is considered too hazardous and unwarranted in the early critical phase. If successful reparative surgery were available, early diagnosis might then be essential and the risk of early bronchoscopy justified.

After the immediate critical phase of the injury has passed and the patient's survival assured, the possibility of bronchial rupture should be considered in all cases of severe chest trauma. The occurrence of a persistent lung collapse is the most suggestive finding and demands further study by bronchoscopy and bronchography. The demonstration thereby of a recent pathologic process in the bronchi, in a previously healthy individual, would serve to establish the diagnosis. When the patient is seen for the first time many months or years after the traumatic episode, the diagnosis of a previous

bronchial injury may not be simple. The symptomatology at this stage is variable and not distinctive. The patient may be entirely asymptomatic. If symptoms are present, exertional dyspnea is the usual complaint, accompanied in some individuals by vague chest discomfort and chronic cough. The physical and X-ray examinations will demonstrate the total lung collapse and suggest underlying bronchial obstruction. This finding, in the absence of bronchopulmonary infection, might be considered diagnostically significant; however, bronchoscopy and lipiodol studies will be indicated in any event to clarify the suspected bronchial pathology. Other and more common causes of bronchial obstruction would, in the final analysis, have to be excluded before accepting the diagnosis of a healed bronchial rupture.

TREATMENT.

The treatment of bronchial injuries is best considered in three phases: Immediate, intermediate and late. The immediate treatment of patients with chest trauma complicated by bronchial rupture would not differ materially from the immediate treatment of any severe chest injury. The early management of severe thoracic injuries to which important contributions were made by military surgeons in World War II has been fully discussed in the recent literature and need not be elaborated upon here. The primary concern is to keep the patient alive by dealing effectively with whatever cardiovascular and pulmonary exigencies arise. Shock, hemorrhage, tension pneumothorax, hemothorax and mediastinal emphysema are the more common urgent complications in cases with bronchial disruption and these are to be treated in the approved manner.

When the early critical phase has been survived and the diagnosis of bronchial rupture established, some form of specific therapy must be given serious consideration. The alternative prospect is the loss of function of one lung. Due to the paucity of cases and the difficulties of early diagnosis, there has been little opportunty for early corrective therapy. Jones and Vinson[2] demonstrated a partial bronchial rupture within about one month of the initial injury, but considered dilatation

of the healing bronchial stenosis inadvisable. Kinsella and Johnsrud[1] established the diagnosis by bronchoscopy within 72 hours of the bronchial injury but attempted no specific therapy. In Case 3 presented above, dilatation of the stenotic area was done on several occasions, but was ineffective in forestalling progression of the bronchial lesion to complete stenosis. Loeffler and Nager[9] treated their case of partial stenosis, the result of a bronchial injury sustained six months before, with repeated bronchoscopic dilatation and electrocoagulation with little apparent benefit.

Operative repair of the bronchial rupture has been mentioned as a possibility by early observers,[10] but has never been attempted in this condition. Sanger[11] did a suture repair on two patients with bronchial lacerations produced by penetrating missiles during the last war, but the end-results are not known. More hope of an effective functional repair would seem to lie in the recent experimental work reported by Daniel[12] and by Longmire,[13] in which tubes of glass, metal or plastic material were used to bridge tracheobronchial defects.

The late management of patients with bronchial injury and its sequelae of partial or complete cicatricial stenosis is of more practical import. The small proportion of cases with residual partial stenosis develop complicating bronchopulmonary infection, as mentioned previously. Ultimate resection would offer the only prospect of complete cure. In the remaining patients with complete bronchial stenosis and chronic lung collapse, treatment to date has been variable and seemingly on uncertain grounds. As previously pointed out, adequate cardiopulmonary studies have as yet not been reported in this group of patients and, lacking these, treatment must remain somewhat obscure. Those who show no clinical evidence of respiratory impairment would appear to have made a satisfactory adjustment and need no active therapy at the moment. Several cases of this type in which the patients remained well over long periods have been described. One of Clerf's[7] patients was followed for eight years and was symptom-free at the last observation. If facilities were available, it might be advisable to have functional studies done in the asymptomatic cases to determine whether there is progressive

distention of the contralateral lung and, if so, to consider measures for limiting the process, to prevent possible future disability.

In the patients with persistent respiratory symptoms, some form of definitive therapy is clearly indicated. If facilities for a complete functional survey are not available, attempts to evaluate attendant cardiopulmonary alterations by simple clinical means should be made. Some estimate of the degree of functional impairment can be gained from the severity of clinical dyspnea. The degree of distention of the contralateral lung can be judged from chest Roentgenograms and its progression, if any, followed. The intrapleural pressures on the involved side should be checked. If the negative intrapleural pressures on the involved side are found unusually high, a therapeutic pneumothorax should be instituted. Its duration is best governed by the clinical progress of the patient. In the case reported by Tyson and Lyle,[3] the symptoms were relieved after several refills of air and the pneumothorax then was discontinued. A follow-up examination a year later revealed normal intrapleural pressures and no recurrence of symptoms. When the negative intrapleural pressures are not found excessive and the cause of symptoms is not readily apparent, a trial period of artificial pneumothorax would still appear justified. In adults, a thoracoplasty to correct mediastinal displacement and limit overdistention of the contralateral lung is to be considered if pneumothorax is not feasible. Pneumonectomy should be reserved for those cases that have definite evidence of infection in the collapsed lung. Since complicating infections are rare in this group with complete stenosis, resection should seldom be required.

CONCLUSIONS.

1. Bronchial rupture is a relatively rare complication of severe, nonpenetrating, external chest trauma. Patients who survive such major injuries are being clinically recognized with increasing frequency.

2. Traumatic bronchial rupture inevitably results in cicatricial bronchial stenosis. In the majority, this stenosis is complete.

3. Pathological sequelae in those with residual complete bronchial stenosis is a chronic atelectatic lung with reduction in respiratory capacity. Unique and noteworthy is the absence of complicating infection in most of these chronic atelectatic states.

4. Diagnosis in the intermediate and late clinical phases is dependent upon a history of severe chest trauma, the discovery of a persistent lung collapse and the bronchoscopic and bronchographic findings of complete bronchial occlusion.

5. In the initial phase of bronchial rupture, treatment of the concomitant severe chest injury is the primary concern. The possibilities of early surgical repair of bronchial disruptions warrant further investigation.

6. Management of old bronchial injuries is handicapped by the lack of basic cardiopulmonary studies. Therapeutic pneumothorax and thoracoplasty are recommended under certain specified conditions. Resection is indicated in the rare case that develops complicating pulmonary infection.

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TURBINATE TREATMENT IN VASOMOTOR RHINITIS.*

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Before a similar meeting of this Society in 1930, Joseph C. Beck read a paper entitled, "Pathology and Intramural Electrocoagulation of the Inferior Turbinate."[1] Fifty patients who had bilateral intumescent and hypertrophic inferior turbinates obtained relief from nasal congestion through submucosal electrocoagulation applied by a single turbinal electrode. Dr. Lee Hurd[2] said in the discussion that for this purpose he had used a twin needle or bipolar electrode for 15 years. A reduction of the hypertrophic turbinate was established with but minimal destruction of the mucosa itself.

In the many papers on vasomotor rhinitis that have been written in the past 18 years, electrocoagulation is but occasionally mentioned and seldom praised. Our President felt that this type of treatment needed review and reconsideration.

Coagulation diathermy of the inferior turbinates plus lateral fracture has given many of my patients with vasomotor rhinitis satisfactory relief of symptoms. Not a cure-all, this is but one of the many forms of therapy that are employed. To define the type of vasomotor rhinitis in which turbinate treatment is indicated, I shall classify the types of therapy that can be used. Second, I shall emphasize the physiology of the inferior turbinate. Lastly, I shall present results of treatment of the inferior turbinate.

Vasomotor rhinitis means a disturbance of the physiology of the nasal lining due to lack of vasomotor control. Excessive sneezing, watery nasal discharge and nasal obstruction are the symptoms. Characteristically, there is an edematous nasal

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mucosa more often pale in color than red or blue. There is mucoid nasal secretion but no pus. The sinuses are clear on transillumination and by X-ray. Many inaccurate synonyms are used to describe this disease. Allergic rhinitis ignores the nonallergic cases. Hyperesthetic rhinitis emphasizes only the sensitivity of the nose. Nervous coryza stresses the neurotic factor seen in but a portion of the group of patients. Paroxysmal sneezing, turgescent rhinorrhea and perennial hay fever are others of the many terms employed to describe those afflicted with a chronic sneezy, stuffy, drippy nose.

Vasomotor rhinitis patients give the drug houses much business. In the search for relief from symptoms, they commonly used drops and sprays as their initial type of therapy. They easily become prey to such medication. Poisoning by privine, for example, is common in this group of individuals. When the rhinologist first sees such a patient, he stops all nasal medication. This is often so beneficial that the rhinologist may feel that he has done all that is necessary.

Of course, the rhinologist looks for infection and obstructive deformities. Sinusitis when present can be alleviated by appropriate measures. An obstructed deviated septum is readily corrected. Polyp removal may completely eradicate the patient's symptoms, but in the majority of the cases no polypi are present and no sinusitis is evident by examination and by X-ray. When no sinusitis is present, sinus surgery is to be deplored. It is this group of people who have caused sinus surgery to fall into disrepute. "I had my sinuses operated on and my nose is just as bad as ever" is a too common complaint.

Allergy is deemed by many doctors to be the true etiologic cause for the patient's symptoms, but the allergic etiology can be overrated. When attacks of a sneezy, stuffy, drippy nose are seasonal, pollens may be blamed. A careful history of the patient's symptoms is most important. When the onset of symptoms coincide with the acquisition of a dog, cat, feather pillow or with the ingestion of certain foods, antiallergic therapy brings dramatic relief. When attacks occur only in certain locations or places, their avoidance can be advocated, but in

the group of patients with chronic vasomotor rhinitis, no good allergic history can be obtained in the majority of cases. Skin tests are negative. The allergist falls back on the theory of "bacterial sensitivity"; he gives vaccines. With elimination diets, he starves the patients. He has to resort to extracts from the vacuum sweeper, and the results of this therapy are often unsatisfactory; thus, unless the allergist can find a good allergen and institute adequate desensitization, other measures are indicated if the patient's symptoms are to be relieved.

Because during pregnancy some women are distressed by increased nasal congestion, the term of rhinitis of pregnancy is used for this type of vasomotor rhinitis. The relation of the nose to the sex organs has long been known. Certain women experience nasal congestion with each menstrual period. Is this, then, a hormonal type of rhinitis? Vasomotor rhinitis may occur at puberty and at menopause. It occurs both in males and in females. It is most common not at but between the ages of puberty and menopause. Careful medical study usually fails to reveal any glandular dyscrasia; therefore, endocrine therapy is not suited for most cases. It is true, of course, that a few cases are cured by hormonal therapy. Equally dramatic cures are achieved when thyroid extract is given when there is a deficiency. Except for the rare case where medical study reveals a need for this type of treatment, it is of no value.

In that lack of vasomotor control seems to be the chief cause for the nasal symptoms of stuffiness, sneezing and watery nasal secretion, the physiologists say that establishment of good vasomotor control is all that is necessary to effect a cure. You need only to take cold showers daily and your vasomotor control will improve. In Boston, we call this "the L Street Brownie" type of treatment. Other authorities, less virile it would seem, advocate warm foot baths nightly to soothe the vasomotor system back to normal and thereby correct nasal complaints. While this type of treatment may be logical, it failed to help any large number of my patients.

The dietary treatment of vasomotor rhinitis has been worked on by biochemists. I have tried only the replacement

of sodium by potassium. In 50 patients I obtained only transitory and slight relief of symptoms. This seemed to be purely psychological and lasted only till the novelty of the treatment wore off.

Psychotherapy helps everyone. Vasomotor rhinitis patients are no exception. Neurotic patients afflicted with this condition stress each symptom. Psychotherapy by the rhinologist

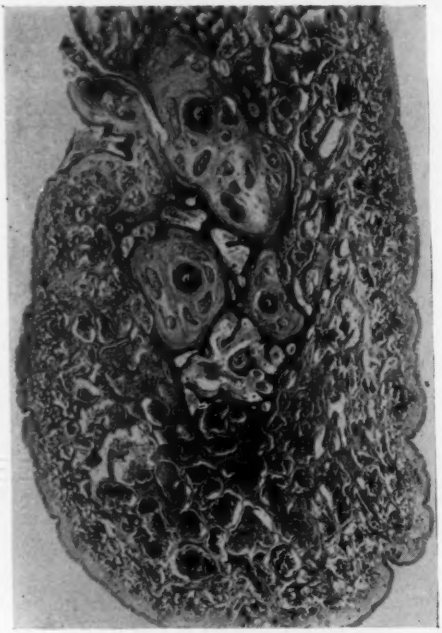

Fig. 1. Normal inferior turbinate.

helps them to a certain extent. A few of my patients have been helped more by psychiatrists than by me. But psychotherapy alone is not the answer to vasomotor rhinitis.

The roentgenologist has held that he could alleviate the stuffy, sneezy, drippy nose of vasomotor rhinitis by small doses of X-ray. Several papers in the literature testify to this,

while an equal number of papers prove that X-ray therapy is of no value. It has failed to help the few patients of mine where it has been employed.

The new antihistaminic drugs afford temporary relief of symptoms. Pyribenzamine seems to work in 60 to 70 per cent of my patients with vasomotor rhinitis. The dosage must be determined for each individual. Some patients are symptom-free on 25 to 50 mg. of pyribenzamine per day. Others require 200 mg. or more per day to remain free of symptoms. Untoward reactions are fortunately uncommon, drowsiness being

Fig. 2. Normal inferior turbinate.

the chief offender. In my hands pyribenzamine produces fewer side reactions than benadryl and is more effective.

Thus it is apparent that there is a large group of patients with vasomotor rhinitis, who do not have sinusitis, who are not benefited by antiallergic therapy, for whom usually no good medical treatment exists, and who desire relief from a stuffy, drippy, sneezy nose.

Fig. 1 shows a cross-section of the inferior turbinate bones. No soft tissue is evident. When clothed with its mucosa, the

inferior turbinate occupies much of the lower half of the nasal airway. When swollen and engorged, the inferior turbinate can fill and block more of the nasal airway than any other structure in the nose. The middle turbinates do their share of obstructing when engorged, but the inferior turbinates are the chief nasal blockers.

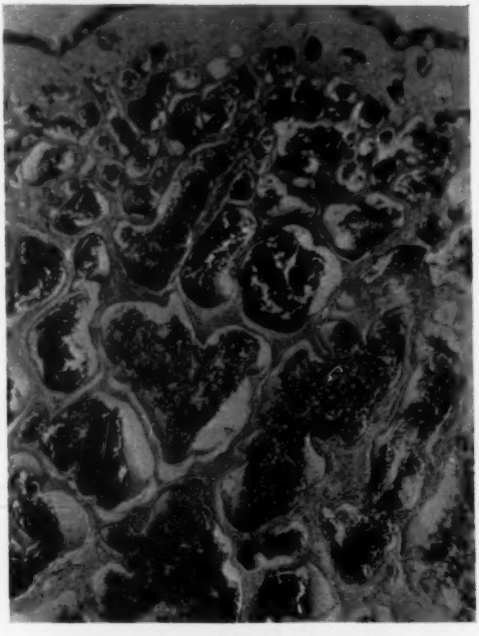

Fig. 3. Normal inferior turbinate.

Fig. 2 demonstrates the blood supply of the inferior turbinate. This is much more profuse than is commonly realized. Primarily for humidification of inspired air, this turbinate requires a generous blood flow.

Fig. 3 shows in cross-section, under high power, the thin-walled large vessels or blood lakes appearing like the radiator

of an automobile. This is true erectile tissue. Like erectile tissue elsewhere, it engorges and distends, providing warmth.

Vasomotor rhinitis may be considered as chronic priapism of the inferior turbinate.

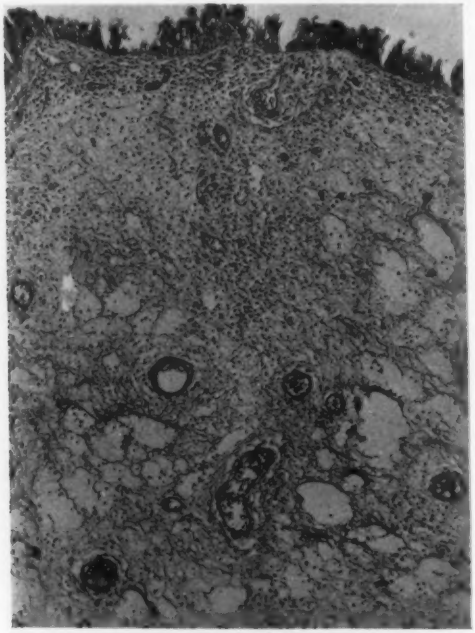

Fig. 4. Vasomotor rhinitis—inferior turbinate.

Fig. 4 shows the edema and cellular infiltration seen in vasomotor rhinitis where there is long continued congestion.

The symptoms of chronic vasomotor rhinitis or priapism of the inferior turbinate can be alleviated by elimination of some of the many cavernous spaces. Elimination of cavernous spaces has been accomplished in many ways. The simplest and

oldest method was to cut off a portion of the inferior turbinate with scissors. This was a bloody procedure, often resulting in adhesions, and either too much or too little turbinate was usually removed. If too much turbinate was removed, crusting and undue dryness resulted in that area of the nose. Parsons, in 1915,[3] described a scarifying operation, using the serrated turbinate knife which bears his name. Dutrow, in 1934,[4] describes the Parsons procedure and stresses the importance of also fracturing the inferior turbinates laterally. His excellent short paper deserves more publicity.

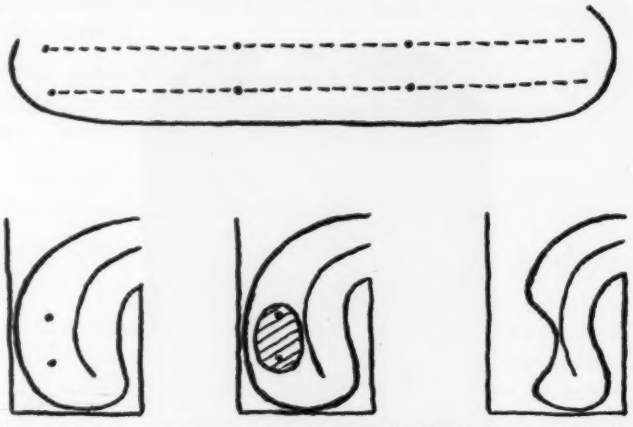

Fig. 5. Diagram of insertion of twin needle electrodes in the inferior turbinate, the area of destruction created, and result obtained.

Various chemicals have been employed on the surface of the turbinates to cauterize more or less deeply. Silver nitrate, trichloracetic acid, resorcinol, phenol and chromic acid are the drugs most commonly used. Injection beneath the surface of sclerosing solutions, such as sodium morrhuate, sodium psylliate and salt solution, have staunch advocates. Certainly cavernous spaces can be destroyed in this fashion. Personally, I cannot control the area to be treated as accurately as I can with the electric turbinate needles. The use of electrocoagulation on the surface and submucosally to destroy portions of the

turbinate is a well established procedure. It is frowned upon only by those who cherish the surface epithelium and the cilia thereon. Applied directly to the surface, considerable destruction can be quickly achieved. This excessive damage to surface epithelium is largely avoided by submucosal insertion of the diathermy needles. The needles should not be placed too close to the turbinate bones. When the electrodes are placed submucosally against the thin inferior turbinate bone and the current is continued too long, necrosis of that portion of the turbinate bone results. In two to four weeks the necrotic portion of bone may slough out. In using electrocoagulation dia-

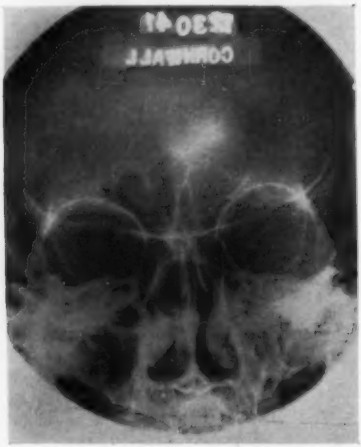

Fig. 6. X-ray demonstrating one inferior turbinate untreated, the other shrunken by submucosal injection of sodium psylliate solution. (Kindness of Dr. R. Swensen, Plymouth, Mass.)

thermy, it is desirable to destroy the least amount of tissue that will produce a satisfactory result. Bone necrosis and epithelial damage are both to be avoided. Several treatments, doing a little at a time, are preferable to doing too much at once.

Under local anesthesia, 10 per cent cocaine solution diluted by an equal amount of 1 per cent ephedrine in salt solution,

the procedure is painless. Twin needles are inserted submucosally for a distance of three-fourths inch at three sites, in the anterior, middle and posterior thirds of the inferior turbinate. The current is applied for a length of time sufficient to cause a white area of destruction as indicated in the diagram. Experiments on a piece of raw meat will establish the time for the length of needle employed at a given setting of the controlling dial. Postoperative discomfort is minimal. A permanent groove is left in the inferior turbinate as indicated. The surface epithelium is but little damaged. Submucosal glands and cavernous spaces are destroyed and replaced by scar tissue.

The following data have been obtained from a review of patients with chronic vasomotor rhinitis seen in private practice. Two hundred consecutive cases were studied.

There were 88 males and 112 females; 44 per cent to 56 per cent indicates a nearly even sex distribution.

The youngest patient treated was 14 and the oldest 72 years of age.

Thirty-seven patients ranged in age from 10 to 19 years.
Fifty-one patients ranged in age from 20 to 29 years.
Fifty-eight patients ranged in age from 30 to 39 years.
Thirty-four patients ranged in age from 40 to 49 years.
Eighteen patients ranged in age from 50 to 59 years.
One patient was in the sixties and another was 72.

More than half of the patients were in the third and fourth decades of life. These figures do not mean that children were not so afflicted, but rather that I did not treat any children's turbinates.

Eighty-nine of these 200 patients had used vasocontrictor nasal drops or sprays excessively, trying to obtain symptomatic relief.

One-sixth of the group, 36 patients, had had a submucous septal resection without relief of nasal obstruction.

Thirty-three patients gave a history of removal of nasal polypi, but had no true polypi present when treatment was instituted. Some patients had polyposis of the mucosa of the middle turbinates.

Twenty patients had some form of sinus surgery previously, but still complained of sneezing, nasal stuffiness and watery nasal discharge. No clinical or X-ray evidence of sinusitis was present when turbinate treatment was started.

One-fifth of the group, 43 patients, had received allergic desensitization treatment without relief of their nasal symptoms.

Many other scattered types of therapy were represented in this group. All therapy employed had been unsuccessful.

The typical complaints of the group were, first, nasal obstruction; second, profuse watery nasal secretion; lastly, excessive sneezing. There were sundry less important complaints.

The nasal mucosa was thick and boggy, it was most commonly pale in color. The turbinates would contrict, but often less readily than normal. There was no pus evident in the nose after shrinking. The secretion was typically mucoid, more often thin and watery than thick and tenacious. Transillumination showed the sinuses to light up well. X-rays were taken in all questionable cases to rule out sinusitis.

The turbinate treatment employed was at first superficial cauterization. This was soon replaced by submucosal electrocoagulation.

The inferior turbinates were treated with trichloracetic acid in 16 patients. Three of the 16 were relieved of symptoms for four years. Two patients returned for help in three years. Ten patients had relief of symptoms for periods of from one to seven months. One patient was not helped at all.

I stopped the use of trichloracetic acid 10 years ago because it did not work well enough.

Twenty-three patients had silver nitrate streaked along the medial aspect of each inferior turbinate. Ten to 50 per cent silver nitrate solution and the fused silver nitrate bead were used. One patient was relieved for nine years, one patient for four years, one for three years and three for one year. The other 17 were helped only for one to six months.

Although silver nitrate occasionally worked well, I abandoned its use because it was usually unsatisfactory.

Thirty patients were treated with a chromic acid bead. This was carefully streaked along the length of the medial surface of the inferior turbinate. I found the results more gratifying.

One patient was symptom-free after six years. Five patients after five years, two patients for four years, one patient returned for another treatment after three years, five patients had relief for two years and wanted the treatment repeated. Six patients had a year symptom-free. The other 10 were relieved only for a period of from three to 12 months.

All of the patients treated with chromic acid experienced considerable reaction. This was immediate and lasted several days. One patient wrote the following poem:

What did you do to my nose?
It acts like a hose;
All day it is flowing,
Soon I must start rowing.
With kleenex all around
It still wets the ground
It's closed, — I can't breathe
Only sneeze
I am trying to smoke —
But I choke,
I'm not able to talk,
Or to sleep, or to walk,
I'm not able to sing,
I just feel like a spring . . .
Have to sit here at home
And attend to the foam,
That's what I must do,
Thanks to you.

While this chap recovered from this reaction and his vasomotor rhinitis symptoms were so benefited that he returned two years later, asking for another treatment; I decided that if I had vasomotor rhinitis myself, I should prefer different treatment.

I have been pleased with diathermy coagulation of the inferior turbinates. The reaction is almost *nil* and the results very satisfactory. Of the 200 cases reviewed, 175 had coagulation diathermy. Six of the 175 patients treated had no good effect. They still complained of their original symptoms. By examination, their airways were improved, but they did not think so. Thus, this treatment is not 100 per cent successful. Of the other 169 patients, one has had no complaints for eight years. One patient has been symptom-free seven years. Another patient has had relief for six years. Three patients have been five years, five patients four years and nine patients three years free of symptoms following treatment. Twenty-six patients have been seen two years after treatment to be symptom-free. Another twenty-six have been found symptom-free one year after treatment. Eight patients came in to have the treatment repeated after two years. The other patients were symptom-free on their first check-up. They were satisfied with the immediate results and agreed to return when their symptoms recurred.

In my office one turbinate is treated. The patient returns four weeks later and the other side is done. A check-up visit occurs four weeks after that. If the result is satisfactory, the patient is advised he need not return until or if the nose again bothers. Unfortunately, this makes a good statistical survey difficult; however, a large enough number of patients have been seen clearly to establish the fact that relief of symptoms usually follows the procedure; and that this relief lasts in terms of years rather than months. I have failed to see any harmful effects. The longest case has been seen eight years after treatment. No atrophy or damage to surface epithelium has been noted.

In half of the 175 cases I have recorded that lateral fracture of the inferior turbinate was carried out. The turbinate can

readily be dislocated outward by pressure with a long-bladed nasal speculum. A sharp "crack" is heard when the turbinate is fractured. No splint or packing is used postoperatively. This simple fracture of the inferior turbinate further increases the nasal airway. In my office it is now a routine adjunct to coagulation diathermy of the inferior turbinate.

CONCLUSION.

Vasomotor rhinitis can be treated adequately only when there exists teamwork between the allergist and the rhinologist. Vasomotor rhinitis does not belong to one specialty alone. Each patient deserves thorough individual study. When destruction of cavernous spaces in the inferior turbinate is indicated, it is easily and simply accomplished under local anesthesia by coagulation diathermy. Postoperative discomfort is minimal. Coagulation diathermy plus lateral fracture of the inferior turbinate obtains satisfactory relief of symptoms in a large majority of patients with vasomotor rhinitis.

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THE NEW TECHNIQUE FOR LOCAL ANESTHESIA IN SINUS SURGERY.*

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Most rhinologists in active practice today appreciate the value of safe and efficient local anesthesia in surgery of the sinuses; likewise, they are cognizant of the unsatisfactory outcome of many sinus operations when anesthesia is inadequate because of its improper administration.

Surgery of all the sinuses can be thoroughly performed with local anesthesia through conduction, and every adult patient undergoing this surgery should be familiarized with its advantages over that of general anesthesia.

All of you, no doubt, are familiar with these advantages; however, for the benefit of current consideration, let us briefly review a few of the more salient factors:

1. With local anesthesia, there is less bleeding and less salivary secretion.
2. Since the throat reflexes are maintained, there is less danger of aspiration of detached fragments of tissue, blood and other secretions, as the operation progresses.
3. Cooperation of the patient is a factor of importance, when a change in the position of the head is required to facilitate operative procedure.
4. There is usually no nausea or vomiting.
5. Surgical shock is less likely to occur, and postoperative convalescence is hastened.

The purpose of this presentation is to convey to you in practical form a simple method of application of conductive

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anesthesia as it relates to surgery of all the nasal accessory sinuses.

Nerve supply of the nose, accessory sinuses and superficial areas requiring surgical consideration are: supraorbital, lacrimal, supratrochlear, infratrochlear, infraorbital, anterior superior alveolar, posterior superior alveolar, anterior ethmoid, posterior ethmoid, sphenopalatine ganglion and maxillary division of the trigeminal nerve.

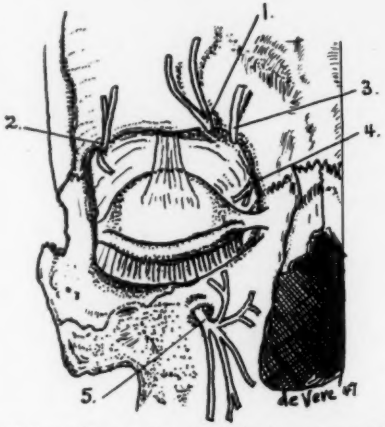

Fig. 1. Terminal branches of the ophthalmic and maxillary divisions of the trigeminal nerves around the orbital ridge.

NERVE BLOCKS FOR ANESTHESIA OF THE FRONTAL, ETHMOID AND SPHENOID REGIONS.

Supraorbital Nerve (see 1, Fig. 1 and Fig. 2 and A, Fig. 4).

Needle employed: 3 cm. long (25 gauge).

Technique: Locate the supraorbital foramen on the margin of the orbital process of the frontal bone. The foramen or notch lies at the junction of the medial third with the lateral two-thirds of the superior orbital ridge. Insert the needle into the skin, immediately over the supraorbital foramen, with the point of the needle directly in contact with the periosteum of the bone in this region.

Solution: 1 cc. of the anesthetic solution will give profound anesthesia in three or four minutes.

Structures anesthetized: Skin, subcutaneous tissue, periosteum and bone of middle third of the eyebrow, the forehead, the upper eyelid and the anterior part of the scalp.

Lacrimal Nerve (see 2, Fig. 1 and Fig. 2 and B, Fig. 4).

Technique: Insert the needle into the skin directly over the superior orbital ridge at a point 2 cm. lateral to the supra-

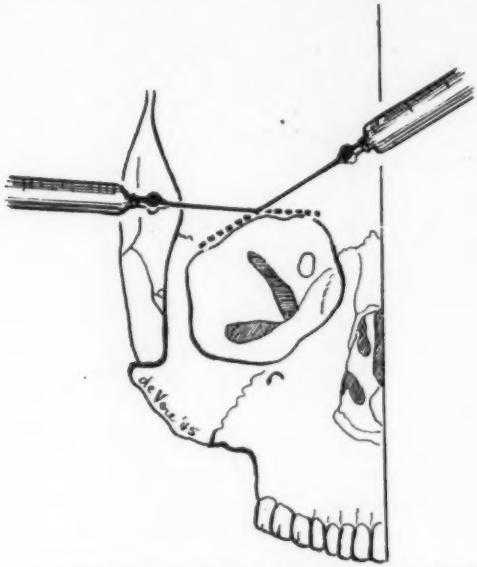

Fig. 2. Alternate injection combining the supraorbital and lacrimal nerve blocks from one central puncture point.

orbital foramen. Direct the needle through soft tissue until it strikes the periosteum and bone of the orbital ridge, at an approximate depth of 5 mm.

Solution: 1 cc. of the solution is sufficient.

Structures anesthetized: Lateral third of the skin, subcutaneous tissue, periosteum and bone of the eyebrow, the forehead, the upper eyelid and the scalp.

Note: An optional injection may be employed, accomplishing both the supraorbital and the lacrimal block from one central puncture point.

Infratrochlear Nerve (see 4, Fig. 1).

The infratrochlear nerve escapes from the orbital cavity below the pulley of the superior oblique muscle, and anesthesia

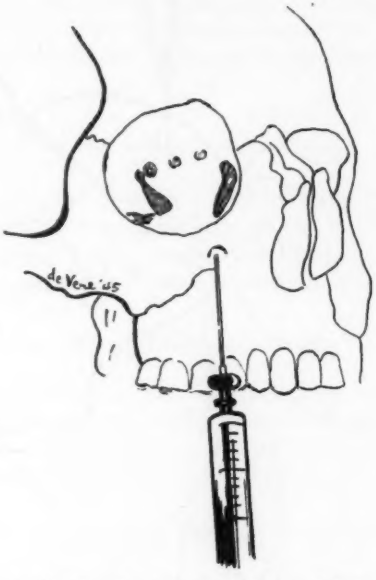

Fig. 3. Intraoral injection for infraorbital nerve block. Note the osteologic relationship of the second bicuspid tooth and the infraorbital foramen.

of this area is accomplished in the preliminary injection of the anterior ethmoid nerve; therefore, independent injection into the infratrochlear nerve need not be considered. The supratrochlear nerve supplies tissues of the medial third of the eyebrow and the forehead, and this nerve is indirectly blocked with the supraorbital injection.

NERVE BLOCKS FOR ANESTHESIA OF THE INFRAORBITAL AND ALVEOLAR REGIONS OF THE MAXILLA.

Infraorbital Nerve (Intraoral Approach) (see 5, Fig. 1 and Fig. 3 and C, Fig. 4).

Needle employed: 3 cm. long (25 gauge).

Fig. 4. Injections for blocking the supraorbital, lacrimal and infraorbital nerves: (A) supraorbital nerve, (B) lacrimal nerve, (C) infraorbital nerve.

Technique: The index finger of the left hand should rest directly over the infraorbital foramen, and the upper lip and the angle of the mouth should be raised with the thumb to expose thoroughly the area which is to receive the needle. Puncture the mucobuccal fold above and lateral to the apex of the upper second bicuspid tooth. Slowly and carefully advance the needle vertically upward to the infraorbital foramen, a distance of approximately 2 cm. The ascending pathway of the needle should not follow the periosteum closely at any point until it strikes the area near the foramen. In other words, the needle bridges the depression of the canine fossa, an important phase of the technique to be considered (Gatewood[1]).

Solution: 2 cc. of the solution is injected.

Structures anesthetized: Skin and subcutaneous tissues of the lower eyelid; skin and cartilage on the lateral side of the nose; subcutaneous tissue, periosteum and bone of the infraorbital region; skin, labial mucosa and gum of the upper lip.

Anterior Superior Alveolar (Dental) Nerve.

The branches from this nerve spring from the infraorbital nerve and traverse the cancellous trabeculae beneath the bony cortex of the maxillary-canine fossa wall; therefore, they may be blocked during the infraorbital injection (de Vere[2]).

Posterior Superior Alveolar (Dental) Nerve (see Figs 5B and 5A).

Technique: Insert the needle in the mucobuccal fold at the apex of the root of the second molar tooth. Push the needle upward, backward and inward for 2 cm. Solution (2 cc.) is deposited, blocking these nerves as they enter minute foramina on the posterior wall of the maxillary sinus.

Structures anesthetized: Mucous membrane and gum of the upper molar region.

MEDIAL ORBITAL INJECTION BLOCKING THE ANTERIOR AND POSTERIOR ETHMOID NERVES.

Anterior Ethmoid Nerve (see Figs. 6B and 6A).

Needle employed: 4 cm. long (25 gauge).

Technique: The puncture point is 1 cm. above the inner canthus of the eyelid. Push the needle through the skin, striking the medial orbital margin, and thence along the medial wall, in close contact with the periosteum, to a depth of 2 cm.

Solution: 1 cc. of the solution is deposited.

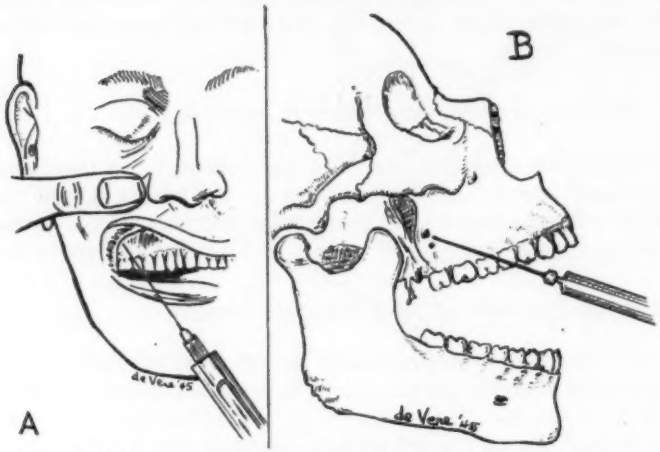

Fig. 5. (A) Intraoral injection of the posterior superior alveolar (dental) nerve. The needle is inserted in the mucobuccal fold at the apex of the second molar tooth and is pushed upward, backward and inward for 2 cm.

(B) Same as (A), showing the osteologic relationship of the molar teeth and the posterior dental nerve foramens on the posterior tuberosity of the maxilla.

Structures anesthetized: Mucous lining membrane of: *1.* the anterior ethmoid cells and the frontal sinus, *2.* the anterior half of the middle and the inferior concha and meatus, *3.* the anterior portion of the cribriform plate of the ethmoid bone, *4.* the anterior and superior parts of the nasal septum.

Posterior Ethmoid Nerve (see Figs. 6B and 6A).

Technique: Advance needle 1 cm. further, posteriorly, along the medial wall of the orbit.

Measurements: The average distance between the anterior and posterior ethmoid foramens is 1 cm. The distance from the medial orbital margin to the anterior ethmoid nerve is

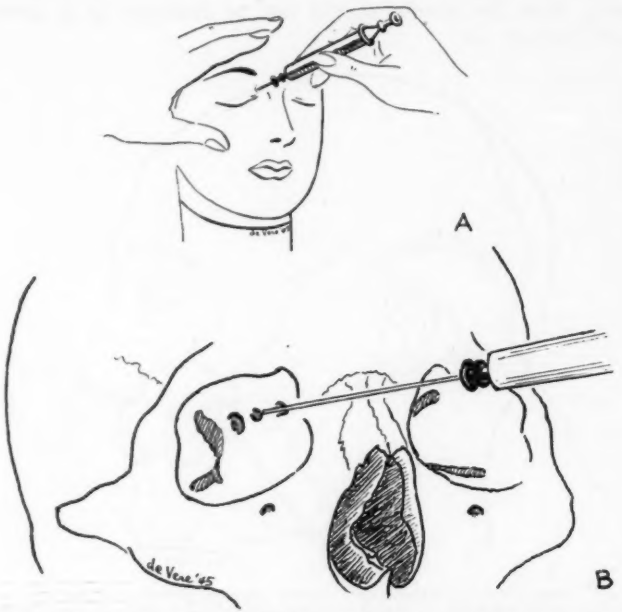

Fig. 6. (A) Medial orbital injection to block the anterior and posterior ethmoid nerves and the supratrochlear and infratrochlear nerves.

(B) Same as (A), showing the osteologic relationship between the foramens of the anterior and posterior ethmoid nerves and the foramen of the optic nerve.

2 cm.; therefore, the total distance over which the needle is inserted to block the posterior ethmoid nerve is 3 cm.

Solution: 1 cc. of the solution is deposited.

Structures anesthetized: 1. Sphenoid sinus and posterior ethmoid cells and *2.* posterior portion of the cribriform plate of the ethmoid bone.

Surgical notes: Great care should be exercised not to insert the needle too far posteriorly, in view of the fact that the optic

nerve is on a direct line with the needle. The average distance between the optic nerve, as it enters the orbit through the optic foramen, and the medial orbital margin is 4 cm.; therefore, it is important, in injection into the posterior ethmoid nerve, that the needle should not be inserted to a greater depth than 3 cm.

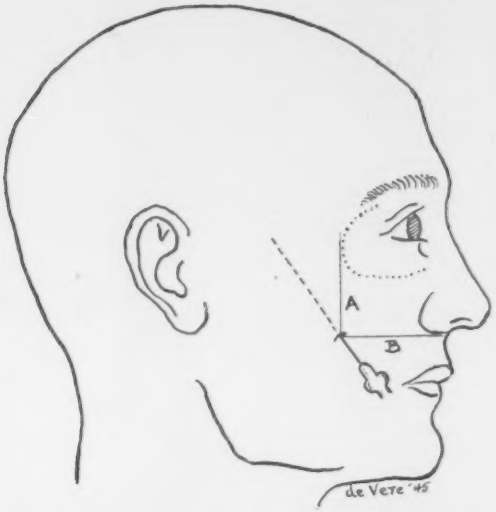

Fig. 7. Extraoral injection for blocking the sphenopalatine ganglion and the maxillary division of the trigeminal nerve. The skin of the cheek is punctured with a needle, detached from the syringe, at a point where two lines intersect: line (A), extending vertically from the lateral orbital rim; line (B), extending laterally from the lower border of the alar cartilage of the nose. This is an alternate injection to supplant injections of the infra-orbital and the anterior and posterior superior alveolar (dental) nerves.

Even if the injection is made 0.5 to 0.75 cm. anterior to the posterior ethmoid foramen, the solution will reach the nerve by infiltration, producing the desired results.

EXTRAORAL INJECTION INTO THE SPHENOPALATINE GANGLION AND THE MAXILLARY DIVISION OF THE TRIGEMINAL NERVE.

Needle employed: 5 cm. long (25 gauge) (see Figs. 7 and 8).

Technique: The syringe is to contain 2 cc. of the anesthetizing solution. The needle, detached from the syringe, is in-

serted in the skin of the cheek at the point where the two lines shown in Fig. 6 intersect. Line "A" extending vertically downward from the lateral orbital rim, line "B" extending laterally from the lower border of the alar cartilage of the nose.

Fig. 8. Same as Fig. 6 (blocking of the sphenopalatine ganglion and the maxillary division of the trigeminal nerve by extraoral injection) showing the osteologic relationship between the ganglion and the nerve trunk to the posterior tuberosity of the maxilla.

Advance the needle upward, inward and slightly backward on the posterior tuberosity of the maxilla, to an approximate depth of 4 cm.

Structures anesthetized: 1. Mucous membrane lining the posterior two-thirds of the nasal fossa and septum, *2.* mucous membrane of maxilla covering the upper gum, and *3.* mucous membrane lining of the maxillary sinus. This injection, if chosen optionally, supplants the injections into the infraorbital and the anterior and posterior superior alveolar (dental) nerves.

Fig. 9. Intraoral injection for blocking the sphenopalatine ganglion and the maxillary division of the trigeminal nerve.

If it is preferred to give the above injection intraorally, the following technique is employed:

INTRAORAL INJECTION INTO THE SPHENOPALATINE GANGLION AND THE MAXILLARY DIVISION OF THE TRIGEMINAL NERVE.

Needle employed: 4 cm. long, 22 gauge; mounted in an extension hub, curved near the tip (see Fig. 9).

Technique: The syringe to contain 2 cc. of the anesthetizing solution.

With the mouth open, puncture the mucous membrane at the highest point between the gum tissue and the inner side of the cheek, above and lateral to the roots of the upper third molar tooth.

Follow the periosteum covering the posterolateral surface of the tuberosity of the maxilla.

Needle is advanced upward, inward and slightly backward to an approximate depth of 3 cm.

Do not allow the needle to assume a perpendicular course, or the needle point will strike the lateral portion of the great wing of the sphenoid bone, and fail to enter the pterygopalatine fossa, across which the maxillary nerve trunk and its ganglion lies. The path of the needle on the posterior tuberosity of the maxilla is anterior to the superior alveolar foramina and vessels.

Blocking the nerves, illustrated in Figs. 1 to 9, is accomplished with injections of a 2 per cent solution of procaine hydrochloride (novocaine) containing solution of epinephrine hydrochloride U.S.P., eight drops to the ounce (0.5 cc. to 30 cc.).

If, however, there is any doubt in the mind of the operator that the technique administered was in any way at variance with that which has been described, it would be well, as a supplementary measure, to make use of topical applications. In addition, the following structures will require supplementary tamponage: the lining membrane of the maxillary sinus, the overlying membrane of the ethmoid capsule and the inferior meatus of the nose. The maxillary sinus is filled with cotton or lint saturated with a 5 per cent solution of a cocaine salt, to which a few drops of solution of epinephrine hydrochloride U.S.P. have been added. This is accomplished during the course of the operation through the opening in the anterior wall. Smaller pieces of cotton or lint, similarly treated with cocaine and epinephrine, are placed in the middle and inferior meatuses of the nose. The cotton or lint is allowed to remain until contact is established as the operation progresses. The piece in the maxillary sinus should remain for five minutes

before proceeding with the removal of the diseased contents of the sinus (Gatewood[3]).

If the above described technique is diligently executed, the lining membranes of all the nasal accessory sinuses will be satisfactorily anesthetized.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of this Society will be held at the Abraham Lincoln Hotel in Springfield, Ill., Nov. 12, 13 and 14. The officers of the Central Illinois Society of Ophthalmology and Otolaryngology are: President, Dr. Clarence E. McClelland, Decatur, Ill.; President-elect, Dr. Clifton T. Turner, Peoria, Ill.; Vice-President, Dr. Harold Watkins, Bloomington, Ill.; Secretary-Treasurer, Dr. Philip R. McGrath, Peoria, Ill.

COMPLICATIONS FOLLOWING TONSILLECTOMY.

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Tonsillectomy fortunately, in the majority of cases, is not followed by any complications. When complications do occur, they may be very serious or even fatal. The medical literature contains records of many different complications that have followed tonsillectomy, but to my knowledge no one has reviewed all the mishaps that have followed tonsillectomy. It is my belief that such a paper is long overdue and the following is an attempt to present such a study. I believe that it is the duty of every physician to report any unusual complication following tonsillectomy, with the expectation that others may profit by his experiences and avoid a similar unpleasant circumstance, or even a possible fatality. I hope, therefore, that additional cases and comments may be added in the future.

Hemorrhage is by far the most frequent and most feared complication of tonsillectomy. Questionnaires sent out by Hill[50] and Loeb[66] revealed a total of 89 deaths due to hemorrhage. Aberrant carotid artery injury was blamed for four fatalities following tonsillectomy, according to Sebileau[97] and Schaeffer.[96] Myerson[78] states: "Death from hemorrhage should not occur in the light of our present knowledge; 77,732 tonsillectomies are listed from three different clinics without a death from hemorrhage." This author is of the opinion that death from hemorrhage following tonsillectomy may in some cases be unavoidable, but the incidence has been greatly reduced over previous years.

Pulmonary complications rank second in importance, consisting of bronchopneumonia, lung abscess, atelectasis, embolism and aspiration of foreign bodies. Postoperative pneumonia and pulmonary abscess follow tonsillectomy more often than is generally believed. Martin[69] reported three

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deaths from bronchopneumonia following tonsillectomy in a series of 14,000 operations.

Moore[76] collected 202 cases of lung abscess following tonsillectomy and estimated that the complication occurred about once in every 2,500 tonsillectomies. In this group of more than 200 postoperative lung abscesses, 39 followed the use of local anesthesia. Kleinert[61] collected over 400 cases of lung abscess following tonsillectomy. The etiology of lung abscess following tonsillectomy is a controversial subject. Some authorities believe that aspiration of blood and infective material during operation is the cause. Jackson[57] believes the abscess is due to septic infarcts and the route is by way of the blood vessels in most cases.

According to Clerf,[17] "Postoperative pulmonary atelectasis is now recognized as a definite clinical entity and with this recognition appropriate treatment has reduced its more serious complications and sequelae to a minimum." Iglauer,[56] Sawyer[93] and others have observed atelectasis following tonsillectomy.

Two cases of pulmonary embolism following tonsillectomy have been reported by Stamberger[102] and Ruedi.[94]

Aspiration of foreign bodies during tonsillectomy is comparatively frequent. Attention is called to a few of the reported cases. Womack[115] reported a case in which a tooth lodged in the left upper bronchus and another tooth in the right middle bronchus following tonsillectomy under gas-ether anesthesia. The teeth were removed by bronchoscopy and normal recovery followed. Jackson[55] reported a case where an enucleated tonsil remained in the left bronchus for seven months without causing a lung abscess. A case of asphyxia following tonsillectomy has been reported by Voorhees.[112] The patient, following a tonsillectomy, developed difficulty in breathing. An emergency tracheotomy was performed. An autopsy revealed the presence of grapefruit seeds in the bifurcation of the trachea. A vistor, against all orders, had given the child grapefruit! The pulp and seeds had been swallowed and, following anesthesia, were regurgitated and inhaled. Asphyxia

might have been prevented if a bronchoscopy had been performed.

Today, with prophylactic measures, the use of sulfonamides and antibiotic drugs, the above group of complications has been markedly reduced.

Deep Cervical Infection Following Tonsillectomy:

Approximately 125 cases of deep cervical infection following tonsillectomy have appeared in the literature.[14,16,18,28,41,42,45,46,49,51,60,64,79,80,92,99,113] The seriousness of this complication is emphasized by 25 fatalities in this group. Two cases of cervical phelgmon with osteomyelitis of the jaw following tonsillectomy are reported by Hochfilzer[51] and Hayden.[45] According to Herman,[49] deep cervical infections following tonsillectomy are due to activation of a latent process in the peritonsillar tissue. Bacteriological study of the pus from these abscesses and the solutions in the mouth and pharynx often reveals the presence of the same organisms in both. The possibility of unsterile solutions as a cause of deep cervical infections is, in my opinion, highly improbable and greatly overemphasized. The prophylactic use of penicillin following tonsillectomy will abort or cure any potential cervical abscess that may have developed. The carotid sinus syndrome may offer some explanation for the numerous sudden deaths that have followed operation of deep cervical abscesses.

Otological Complications:

Acute otitis media and mastoiditis have followed tonsillectomy.

Intracranial Complications:

Intracranial complications following tonsillectomy are extremely rare, but when they do occur are usually fatal. They consist of lateral sinus thrombosis, cavernous sinus thrombosis, meningitis, cerebral abscess and embolism. Hara and Courville,[40] in a survey of the literature from 1900 to 1940, found 76 cases of intracranial complications of tonsillar origin, but only 22 of these followed tonsillectomy. Of these, 16 cases were fatal. Campbell,[13] Cadbury and Siddal,[12] Comer,[19]

Dean,[26] Goodman,[37] Harris and Yenikomshian,[44] Ide,[55] Keen,[60] Loeb,[66] Nesbitt,[79] Tumpeer and Levinson,[108] and Zambrini and Montanaro[117] have also written on this subject.

Infectious Diseases Following Tonsillectomy:

Measles, mumps, chicken pox, scarlet fever, diphtheria, meningitis, encephalitis and poliomyelitis have been attributed to tonsillectomy. With the exception of poliomyelitis, it is generally conceded that the infection existed prior to the operation. According to Pedersen,[84] from 1910 to 1946, no less than 314 cases of poliomyelitis following tonsillectomy have been reported. There seems to be conclusive evidence that the bulbar type of poliomyelitis occurs more frequently after tonsillectomy. Cunning[20] found poliomyelitis following tonsillectomy relatively infrequent. He reviewed 11,204 tonsillectomy patients over a seven-year period and found only four cases of poliomyelitis following tonsillectomy. The widespread alarm on the part of the public, and shared by doctors in some communities, is unfounded according to his statistics. In the author's opinion, tonsillectomy need not be postponed during the summer and fall months unless there is a known case of poliomyelitis in the community.

Subcutaneous Emphysema Following Tonsillectomy:

The incidence of subcutaneous emphysema following tonsillectomy is rare: only 17 cases have been reported.[15,27,59,60,67,74,82,86,88,91,93,104,111] It has occurred after local as well as general anesthesia in both children and adults. Its appearance may be alarming to both the surgeon and patient, although no permanent serious results have been recorded.

Drug Idiosyncrasy Following Tonsillectomy:

Hypersensitivity to aspirin, morphine, atropine, phenobarbital, aminopyrine, novocaine, adrenalin, pontocaine and cocaine has followed tonsillectomy.[52] Individuals who are subject to any drug idiosyncrasy should be acquainted with the fact, so that they may call it to the attention of other physicians. An inquiry relative to this subject before the operation may prevent an embarrassing situation. Several cases of sensitivity

to novocaine following tonsillectomy have been reported. Mayer[72] reported 43 deaths following the use of various local anesthetics. It is a wise precaution to color all cocaine solutions so that they may not be accidentally injected in place of novocaine.[43,48,70,71,94,100,110]

Bacteremia Following Tonsillectomy:

Van Eyck[109] found a transitory bacteremia present in 40 out of 100 examined cases, where blood cultures were taken right after tonsillectomy. The following micro-organisms were found in the blood: Gaffkya, tetragena, pneumococcus, hemophilus influenzae, cornybacterium pseudodiphtheriae, streptococcus hemolyticus, streptococcus viridans, Neisseria catarrhalis, staphylococcus aureus and others. These findings confirm other reports on transitory bacteremia following tonsillectomy.[4,32,39,68,77,90] Acute exacerbations of polyarthritis, nephritis and endocarditis which often follow tonsillectomy might be explained on this basis (Curtis[22]).

Agranulocytosis Following Tonsillectomy:

Three cases of fatal agranulocytosis following tonsillectomy have been reported by Reich[85] and Seiferth.[98] The importance of preoperative leucocyte count is stressed. There is a possibility that the acute phase of this condition may be initiated by tonsillectomy.

Thrombophlebitis of the Leg Following Tonsillectomy:

Thrombophlebitis of the leg following tonsillectomy is very rare. Harlowe reports such a case.[43] A white male, aged 21 years, underwent a local tonsillectomy by the sharp dissection and snare method. Some difficulty was encountered because of the large size of the tonsils. The postoperative course was uneventful until the fourth day, when the patient suddenly developed a temperature of 103°. The next day he complained of pain and tenderness in his leg, and a tentative diagnosis of deep femoral thrombophlebitis was made. During the next nine months of treatment there was no marked improvement in the condition of the leg.

Virus Infection Subsequent to Tonsillectomy:

A 16-year-old girl developed a bulbar type of poliomyelitis following tonsillectomy, and died 16 days later. In a second patient, 24 years old, three days after operation a typical myringitis bullosa hemorrhagica developed in the left eardrum. The patient at first could hear a whispered voice at a distance of three-fourths meter, then the hearing gradually became impaired until a whispered voice was heard only ad concham. There was a coarse horizontal nystagmus and rotary nystagmus of the first degree to the left and an occasional nystagmus to the right. One week after the onset of symptoms, the patient's condition returned to normal. In both cases all bacteriological studies were negative. A virus infection was believed to be the cause of the disease in both cases.[9]

Excessive Scarring of the Pharynx After Tonsillectomy:

Excessive scar formation occasionally follows tonsillectomy. The majority of these cases are due to unskilled operators and faulty technique. Individual peculiarities in healing may account for a few of these cases. By careful dissection and unhurried operations, the majority of these undesirable results may be avoided.

Erysipelas of the Oral Mucosa Following Tonsillectomy:

Sprenger[101] reported 10 cases of erysipelas of the oral mucosa following tonsillectomy. He observed these cases during a six-year period among 2,290 tonsillectomies. According to him, three other similar cases have been reported in the literature by Buchband,[10] Laemmle[63] and Miller.[75] Many of these infections occurred postoperatively as a result of the patients eating unclean fruit that had not been peeled. Thus by injuring the granulations in the tonsillar fossa an infection was incurred, supposedly by streptococci. Rapid recovery followed the use of sulfonamides and conservative therapy.

Hemiplegia Following Tonsillectomy:

A general tonsillectomy was performed on a 19-year-old male. During the operation the patient came out of the anesthetic once and coughed considerably. Seven days later he

developed a left-sided paralysis. Several convulsions occurred during the next two days and death followed on the tenth day after tonsillectomy. According to Gracey,[38] who reported the case, an embolism occurred in the motor area with a resulting left hemiplegia.

Status Thymicolymphaticus Following Tonsillectomy:

Considerable controversy has been written in regard to the existence of such an entity.[1,7,23,29,34,79,105] The fact cannot be denied that sudden death does occasionally occur during general tonsillectomy without an adequate explanation. I believe that many of the so-called thymic deaths are the result of other causes. For some undetermined reason certain children will not tolerate what we might assume to be the normal amount of anesthesia and surgery during tonsillectomy.

Bilateral Gangrene of the Feet Following Tonsillectomy:

A unique case of bilateral gangrene of the feet following tonsillectomy was reported by Wright.[116] A girl two years of age developed bilateral gangrene of the feet 17 days after tonsillectomy. Six weeks later the areas of gangrene had cleared up, but the third toe of the right foot had sloughed off.

Cephalic Tetanus Following Tonsillectomy:

One case of cephalic tetanus following tonsillectomy and adenoidectomy has been reported.[54] The patient, a boy nine years of age, developed typical symptoms of tetanus, 13 days after operation. The diagnosis was confirmed by inoculation of experimental animals. Complete recovery followed the use of tetanus antitoxin.

Subluxation of the Atlas Following Tonsillectomy:

A case of subluxation of the atlas following tonsillectomy was reported by Friedberg.[36] The patient, an 11-year-old colored girl, was admitted for a tonsillectomy and adenoidectomy. Physical and laboratory findings were normal on admission. No difficulties were encountered during the operation and the patient was discharged the next morning.

The patient returned 12 days later, at which time the tonsil fossae appeared to be healing satisfactorily; however, she complained of tenderness in the right anterior cervical region and pain upon rotation of the head. There was some tenderness along the sternocleidomastoid muscle with moderate adenopathy.

Twenty-six days postoperatively, a torticollis to the right was observed. The head was held more or less rigidly, and only a very slight degree of flexion, extension and rotation to the right was possible. No rotation to the left could be produced and manual attempts resulted in pain and rotation to the lower spine. She was unable to lie down with comfort. Marked spasm and tenderness of the posterior cervical muscles was noted. The spinous process of the second cervical vertebra was prominent to palpation.

Roentgen ray examination of the atlantoaxial joint revealed an absence of the joint space between the lateral facets (left) of the atlas and the axis. Such X-ray findings were felt to be pathognomonic of atlantoaxial dislocation or subluxation by the orthopedic consultant who was asked to see the patient.[5]

Hospital treatment consisted of hyperextension on a Bradford frame for 24 hours. Head traction was then applied, beginning with one pound and increasing to two pounds, as tolerated. The traction was removed after 13 days, but the Bradford frame was continued for five more days. A normal recovery followed.

The orthopedic consultant felt that there had been no trauma and that the subluxation was secondary to infection in the upper respiratory tract, causing arthritis of the atlantoaxial joint.[6]

Styloid Ossification and Injury of the IXth Nerve After Tonsillectomy:

Fowler[35] reported a case of bilateral styloid ossification and injury to the IXth nerve following tonsillectomy. The patient, a 33-year-old male, complained of a constant and marked discomfort in the throat and an unpleasant taste.

Ossification of the styloid process is of infrequent occurrence and may be unilateral or bilateral. The tonsil usually appears shallower than normal. The anomaly is often discovered accidentally while injecting the local anesthetic. By careful dissection the tonsil may be easily removed without harming the styloid process. One should never attempt to amputate a styloid process with a tonsil snare.

Hyperpyrexia Following Tonsillectomy:

Several cases of hyperpyrexia following tonsillectomy have been reported.[24,83,87,114] Temperature elevations of 107.4° F. after tonsillectomy have been recorded before death. No cause for death was found in the cases where autopsy was performed.

Air Embolism Following Tonsillectomy:

A case of air embolism following tonsillectomy has been reported by Steinberg.[103] A tonsillectomy was performed on a male 18 years of age under a general anesthesia. During the operation approximately 200 cc. of air were sucked into the cervical veins and then trapped in the right side of the heart, where it remain for several days. Complete recovery occurred in five days following oxygen therapy.

Triplegia Following Tonsillectomy:

An unusual case of triplegia following tonsillectomy has been reported by Brahdy.[8] Five days following operation, an eight-year-old boy developed what was first diagnosed as poliomyelitis, but later changed to triplegia. According to the author, multiple emboli were the causative factor. A complete flaccid paralysis and anesthesia below the sixth thoracic vertebra was still present three and one-half years after tonsillectomy.

Mediastinal Abscess Following Tonsillectomy:

One case of mediastinal abscess following tonsillectomy has been reported (Tremble and Hewitt[107]). The operation was performed under avertin and amylene hydrate gas anesthesia.

The diagnosis was made on the fourteenth postoperative day with barium solution aid fluoroscopy. A normal recovery followed after 36 days.

Acute Coronary Occlusion Following Tonsillectomy:

One case, in the author's experience, developed an acute coronary occlusion following injection for local tonsillectomy. The autopsy confirmed the diagnosis and revealed an occlusion of the left coronary artery. One other identical case has since been brought to his attention.

Severe Gastric Hemorrhage Following Tonsillectomy:

Two cases of severe gastric hemorrhage following tonsillectomy have been reported by Bunker.[11] The first case, a male, aged 27 years, underwent a local tonsillectomy. The operation was performed in the morning, and late that afternoon he had a large bowel movement, at which time he passed approximately 400 cc. of bright red blood. Careful examination of the tonsil fossa revealed no sign of bleeding to explain this hemorrhage in the gastrointestinal tract. The bleeding from the bowel continued for several days and was severe enough to require several blood transfusions. Complete laboratory studies were negative. Subsequent gastrointestinal studies revealed no ulcer or neoplasm to account for this bleeding. A diagnosis of acute gastritis was believed to account for the hemorrhage. The patient operated a tavern and it may have been that he imbibed too much of his own products and caused this condition. A normal recovery followed.

The second patient gave a history of occasional gastric distress. Tonsillectomy was performed without difficulty under local anesthesia and shortly afterwards he began to vomit large amounts of bright red blood. The tonsil fossa revealed no sign of bleeding to account for this hemorrhage. He continued to vomit for 24 hours, after which he subsided under the usual treatment of morphine, ice, etc. Later gastrointestinal studies revealed a definite gastric ulcer which was the source of the bleeding. A normal recovery followed.

Anesthesia Deaths Following Tonsillectomy:

Nothing is more discouraging or as tragic as a death on the operating table. The exact number of such fatalities which have occurred is unknown. What part the anesthetist, the patient and the surgeon play in these mishaps is often difficult to determine. Tonsil anesthesia is one of the most difficult to give properly. One can always give the patient more anesthesia if it is too light. It is a wise precaution to follow the patient's progress during the operation so as to recognize any danger signals before it is too late. It is unwise to continue the operation in the face of alarming or dangerous symptoms. Immediate postoperative attention following a general anesthetic is of utmost importance.

Multiple Complications Following Tonsillectomy:

Several complications may occur in one patient following tonsillectomy. One, two, three or even more have occurred. The first complication may lead to others, or they may occur independently of each other. Prevention of the first may forestall all others.

Complications may arise after tonsillectomy, just as they do after any other type of operation. It is, therefore, very important for every otolaryngologist to have a comprehensive knowledge of all the various mishaps that may follow tonsillectomy, classify these complications, study their causes and discuss means of preventing them.

SUMMARY.

1. Complications may arise after tonsillectomy, as they do after any other type of operation.

2. A careful physical examination and thorough laboratory study before tonsillectomy will minimize the operative and postoperative complications.

3. The use of sulfonamides and antibiotics has reduced many of the complications that were prevalent in the past.

4. It is important for every otolaryngologist to have a comprehensive knowledge of all the various complications that may follow tonsillectomy.

COMPLICATIONS FOLLOWING TONSILLECTOMY.

(Type of Complication)	No. Reported Cases	No. Reported Deaths
1. Hemorrhage:		
1. Primary hemorrhage. Immediately or within six hours after operation.		
2. Secondary hemorrhage. From six hours to two days following operation.		
3. Delayed hemorrhage. Occurring two to 20 days postoperatively.		
4. Fatal hemorrhage.		
a. Excessive bleeding following primary, secondary or delayed hemorrhage.		
b. Blood dyscrasias, *i.e.*, hemophilia, severe secondary anemia, pernicious anemia, purpura, leucemias and Hodgkin's disease.		
c. Severe hypertension and advanced arteriosclerosis.		
d. Aberrant vessels, *i.e.*, tortuous internal carotid artery.		
e. Unskilled operator.		
	?	Over 100
2. Pulmonary Complications:		
1. Bronchopneumonia.	?	?
2. Lung abscess.	Over 400	?
3. Atelectasis.	6	?
4. Embolism.	4	?
5. Aspiration of foreign bodies.	?	?
3. Deep Cervical Infections:	Over 125	25
4. Otological Complications:		
1. Acute otitis media.	?	?
2. Mastoiditis.	?	?
5. Intracranial Complications:		
1. Lateral sinus thrombosis.	4	?
2. Cavernous sinus thrombosis.	7	6
3. Meningitis.	4	3
4. Cerebral abscess.	2	2
5. Embolism.	5	5
	22	16
6. Infectious Diseases:		
1. Poliomyelitis.	314	35?
7. Subcutaneous Emphysema:	17	0
8. Drug Idiosyncrasy:	?	12?
9. Bacteremia:	?	?
10. Agranulocytosis:	3	3
11. Thrombophlebitis of the Leg:	1	0
12. Virus Infection:	?	?
13. Excessive Scarring of the Pharynx:	?	?
14. Status Thymicolyphaticus:	100?	100?
15. Erysipelas of the Oral Mucosa:	13	0
16. Hemiplegia:	1	1
17. Bilateral Gangrene of the Feet:	1	0
18. Cephalic Tetanus:	1	0
19. Subluxation of the Atlas:	1	0
20. Styloid Ossification and Injury of the IXth Nerve:	1	0
21. Hyperpyrexia:	4	3
22. Air Embolism:	1	0
23. Triplegia:	1	0
24. Acute Coronary Occlusion:	2	2
25. Mediastinal Abscess:	1	0
26. Gastric Hemorrhage:	2	0
27. Anesthesia Deaths:	?	?
28. Multiple Complications:	?	?

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Garberson Clinic.

THE CELLULAR AND HUMORAL FACTORS INFLUENCING CONSTITUTIONAL RESISTANCE AND THE DEVELOPMENT AND CONTROL OF LOCAL LESIONS.*

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Columbus, Ohio.

Every physician, irrespective of his special interest, is challenged at times by the patient who does not respond as expected to the procedures that have been used in correcting what, at first, appeared to be a relatively simple difficulty. Members of the otolaryngologic specialty in this community have learned to investigate carefully the constitutional resistance and to evaluate any acquired susceptibility of a given patient before advising any surgical procedure. Thus, it has been possible many times, for the specialist working with the internist and hematologist to avoid dangerous complications by anticipating and preventing them in advance.

The rôle of infection, latent or actual, must always be meticulously considered. Ordinarily influenza is a mild, self-limited disease, but during the winters of 1917 and 1918, it assumed epidemic proportions and was complicated by a deadly secondary invader, the hemolytic streptococcus, which profoundly influenced both the morbidity and the mortality among civilian and military populations alike. The factors responsibile for the high mortality were not known at that time. Speculation pointed to highly virulent organisms, plus mass mobilization, but it remained for carefully controlled experimental studies in the laboratory to analyze and specifically reveal the several important factors involved.

Human influenza virus A, when introduced into the undamaged, intact nasal passages of the nutritionally normal maca-

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cas rhesus monkey, will invade the tissues and produce a mild, transitory disease syndrome, characterized by a profound neutropenic leucopenia (see Fig. 1). On or about the ninth day, coincident with the appearance of neutralizing antibodies, the prepiheral white blood count returns to normal.[1] This is a cellular phenomenon common to all virus diseases.

The intranasal introduction, into normal monkeys, of virulent hemolytic streptococci, Lancefield strain C, isolated origi-

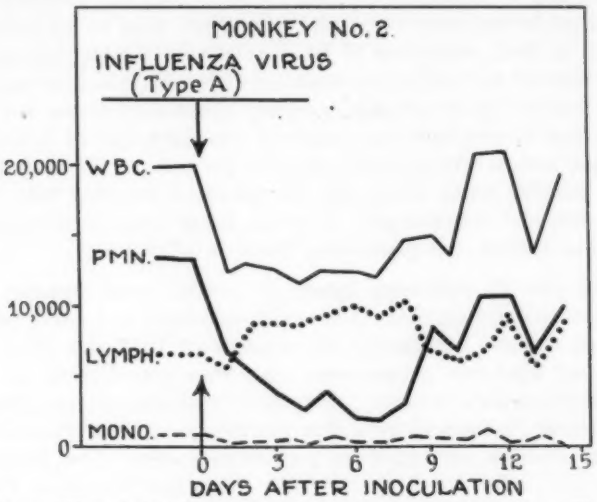

Fig. 1. Virus induced (intranasal) leucopenia with recovery. Normal macacus rhesus.

nally from a human patient with scarlet fever, is followed promptly by invasion, but in this instance a marked polymorphonuclear leucocytosis is elicited, and this cellular response on succeeding days, reflects the degree of success attained by the animal host in controlling the invasion (see Fig. 2).[2]

Since virus apparently inhibits specifically those cellular elements required to combat streptococci successfully, it is not perhaps too unexpected for a monkey (or man), who is exposed to the latter while still experiencing the virus induced

leukopenia, to fail to mobilize the neutrophilic granulocytes and, therefore, sustain a much more serious overwhelming invasion of streptococci, with, on occasion, a terminal, fatal septicemia (see Fig. 3).

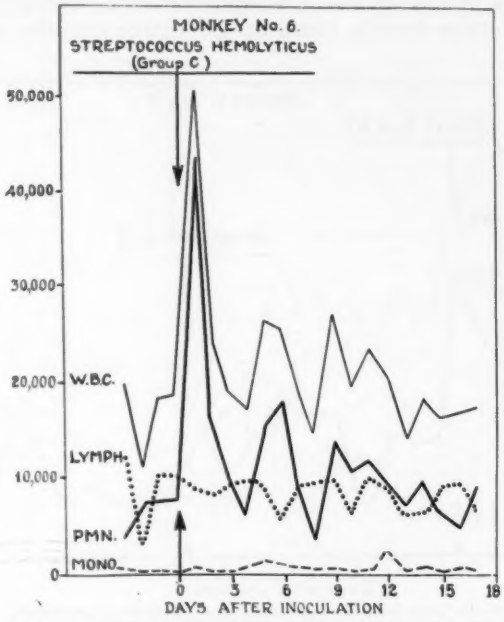

Fig. 2. Hemolytic streptococcus induced polymorphonuclear leucocytosis with recovery. Normal macacus rhesus.

The initial infection with a specific strain of streptococcus always elicits a high individual leucocyte response, probably because the opsonocytophagic index is low; *i.e.*, the ability of each polymorphonuclear neutrophil to phagocytize streptococci is very low; however, reinfection with the same strain of organisms two or three months later is equally well combated without any rise in the total white count.[3] The opsonic index in this instance becomes very high immediately and the efficiency of the cellular defense rises correspondingly, as many

as 40 cocci being counted within one polymorphonuclear granulocyte. If, then, influenza virus enters the picture, the opsonic index falls to zero within 24 hours. With both the quantity and the phagocytic function of the granulocytes effectively curtailed by the virus, the hemolytic streptococci proceed to multiply within the host and signs and symptoms of progressive infection develop (see Fig. 4). Either quantity or humo-

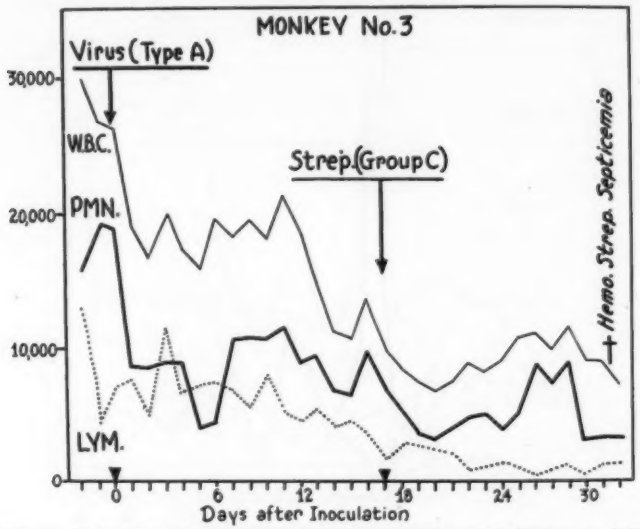

Fig. 3. Hemolytic streptococcus infection superimposed during an influenza virus induced leucopenia with terminal fatal septicemia without benefit of adequate cellular defense.

rally induced quality in the neutrophils seems essential to streptococcus control. Experimental reinfection with hemolytic streptococci occasionally results in what we have interpreted as an acute glomerular nephritis secondary to a virus induced leucopenic episode and presumably on an allergic basis (see Fig. 4).

Thus far we have been detailing responses observed in macacus rhesus monkeys under optimal nutritional conditions and under environmental circumstances presumed to be those

prevailing for normal adult human individuals in similar conditions. We now desired to test the influence of certain dietary restrictions on the cellular-humoral immunity, natural and acquired, of our monkeys. Following the earlier reports of Langston and his associates,[4] we started withholding various fractions of the vitamin B complex and studying the bone

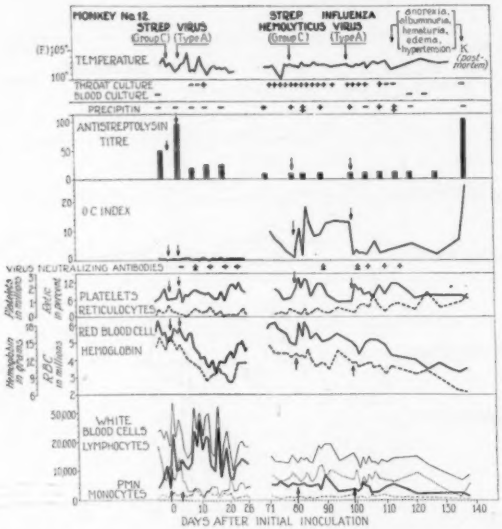

Fig. 4. Reinfection with hemolytic streptococci of the original strain elicited a high opsonic index without repeating the initial absolute leucocytosis to bring about recovery. When, however, influenza virus infection was superimposed, the opsonic index suddenly fell, granulocytosis was inhibited, the streptococci multiplied and invaded progressively, and, in this instance, an acute glomerular nephritic syndrome resulted.

that both yeast and liver contained essential food factors which could prevent the progressive marrow hypoplasia which marrow and blood cell responses. It soon became apparent developed when a strict vitamin B deficiency was maintained in an otherwise adequate diet for from 60 to 90 days (see Fig. 5). This hematopoietic essential common to both liver and yeast proved eventually to be folic acid, pyteroylglutamic acid.

In the macacus rhesus monkeys on a low folic acid intake with a progressive leucopenia, both influenza virus and hemolytic streptococcus infections proved far more serious than when similar exposures were given to their nutritionally normal colony mates.[3] Humoral antibodies developed similarly both in time and titer, but an abortive attempt only was possible on the part of the bone marrow in the folic acid deficient

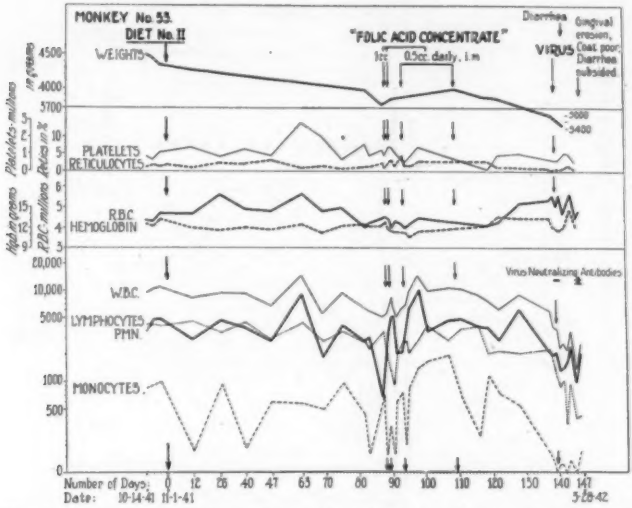

Fig. 5. Folic acid deficient diet produced progressive panmarrow hypoplasia — temporarily relieved by a small supplement of this essential vitamin fraction — relapsing with recurrence of the leucopenia in the absence of folic acid; this animal succumbed to intranasal influenza virus infection.

animals and terminal septicemia almost invariably developed, associated with a more profound terminal neutropenic leucopenia (see Figs. 5 and 6).

As the neutropenia develops in these animals, local lesions begin to appear, particularly about the face, nose and mouth—conjunctivitis, nasopharyngeal, lip and gum ulcerations, erysipelas of the face and, not infrequently, intestinal dysentery. With the giving of adequate supplements of folic acid, the

cellular defenses immediately increase, and prompt spontaneous disappearance of the lesions occurs.

The increased susceptibility to both virus and coccus infections of monkeys, leucopenic on a nutritional deficiency basis,

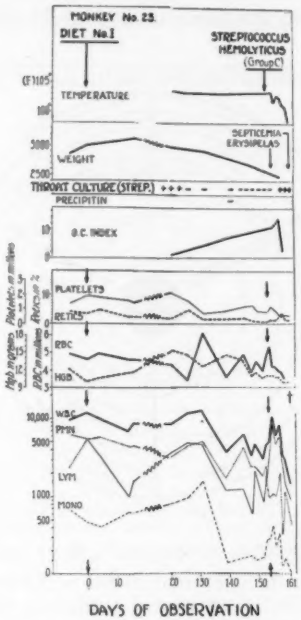

Fig. 6. Hemolytic streptococci introduced intranasally in folic acid deficient, leucopenic monkey resulted in a fatal septicemia, with terminal, profound leucopenia.

is statistically significant. Intranasal instillation of influenza virus A produced evidences of a self-limited disease with transitory leucopenia and the development of effective protective neutralizing antibodies in 27 normal, nutritionally healthy rhesus monkeys, without a single fatality. Eight folic acid deficient monkeys with panmarrow hypoplasia and neutropenic leucopenia, when given the same intranasal instillation of influenza virus A, succumbed in from two to 11 days,

with a fatal pneumonitis, from which the virus was recovered on culture postmortem. When hemolytic streptococci, strain C, were instilled into the nares of 32 normal rhesus monkeys, invariably a prompt neutrophil granulocytosis occurred and recovery was complete in all but two instances in which death followed the initial infections at 15 and 21 days, respectively. In sharp contrast, the same strain of streptococci produced a

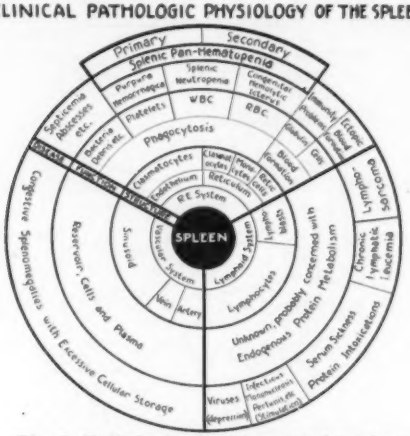

Fig. 7. Pathologic physiology of the spleen.

fatal septicemia within seven to 13 days in five or six nutritionally deficient, leucopenic monkeys. Coincidental humoral antibody studies failed to reveal sufficient differences in time of appearance or titer to significantly affect the variations in susceptibility as noted.

Clinical studies, which parallel the rhesus observations and which have been carried on in our clinic for a number of years, fully confirm the very great significance of the cellular factors in the resistance and susceptibility of the human individual for both intrinsic and extrinsic sources of infection, and, therefore, of the importance which attaches to this basic defense mehanism in any surgical or medical condition.

With this fundamental premise in mind, may I call your

attention to another mechanism which is assuming increasing importance, as a factor for consideration in the causation of clinical syndromes in which some disturbance in the circulatory blood cell equilibrium is apparent. Emphasis on the integrity of the bone marrow, as the source of red cells, granulocytes and platelets, has been given in the preceding examples of virus and coccal infections, and on the impor-

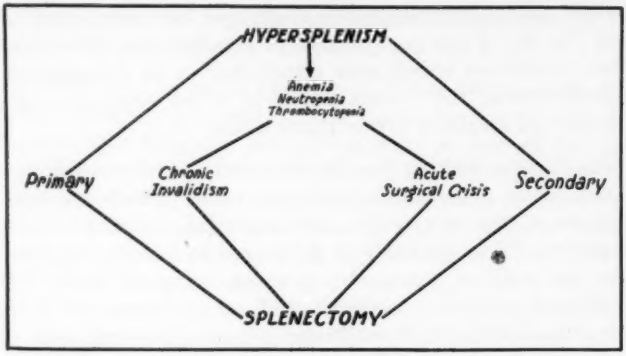

Fig. 8. Hypersplenism.

tance of an optimum diet to a maximum cellular defense mobilization. An equally important rôle is played by the spleen, in its physiologic functions as a reservoir for blood cells and as a conservator of and storage depot for the basic materials from which the continuous new supply of blood cells must come.

It has become abundantly evident in recent years that this organ (Fig. 7) which for so long was considered a functional enigma to physiologist and clinician alike may inherit an instability which can result in the dangerous withdrawal and pathologic destruction of any or all of the essential circulatory elements of the blood — and this despite an heroic effort on the part of all the bone marrow of the body to compensate with a maximum cellular production. A wide range of clinical syndromes and diagnostic entities may now be explained upon the basis of this mechanism, which we have described as

hypersplenism (see Fig. 8).[5] Congenital hemolytic icterus,[6] thrombocytopenic purpura hemorrhagica,[7] primary splenic neutropenia[8] and primary or secondary splenic panhematopenia[5] are included within this mechanistic concept. An adequate adrenalin test, reflecting specific cellular hypersequestration and a carefully correlated analysis of the cells of the bone marrow, both qualitative and quantitative, form the objective bases for diagnosis. Splenectomy is the specific remedy when a hypersplenic syndrome has been established (see Fig. 8). Prior to the etiologic treatment or correction of other conditions which may complicate or be complicated by hypersplenism, the re-establishment of an adequate cellular equilibrium assumes prime importance.

The factors making for the successful practice of otolaryngology, or of any medical specialty, must include the responsiveness of the nonspecific, but essential, cellular blood and connective tissue elements as influenced by heredity and nutrition, as well as diagnostic acumen, surgical skill, and a knowledge of the specificity and effectiveness of modern chemotherapeutic and antibiotic drugs. Utilizing and combining the accumulated wisdom and experience, which is resulting in the recognition of man as a united, integrated, psychosomatic whole — composed of many highly specialized parts, functioning effectively for the most part, however, in a hostile environment — the modern physician may achieve both preventive and curative triumphs unimagined even a few short years ago.

DISCUSSION.

The question of the interpretation of the blood picture in infancy and childhood has been raised by Dr. Beatty. This is particularly important to you here because of the large demand of the younger age group upon the otolaryngorhinological specialist. The differential white blood cell formula which characterizes the normal adult is not attained until the tenth to the fifteenth year. The granulocyte-lymphocyte ratio of maturity is reversed in infancy and only gradually assumes the 70:30 relationship with adolescence. Be cautious, there-

fore, in diagnosing a pathologic lymphocytosis or abnormal neutropenia in childhood. Hematopoiesis is less stable, and nutrition more readily deficient in infancy and childhood, infections are more common, and the magnitude and suddenness of cellular disequilibria are correspondingly greater and less significant. Qualitative changes in the blood cells assume greater significance under these circumstances and must be critically evaluated. Hemoclastic crises on a hypersplenic basis may occur at any age and whether recognized at two weeks or 80 years of age, splenectomy is indicated.

The fact that proteins and the essential amino acids were not specifically mentioned in this discussion previously does not mean that they are not as necessary as any of the vitamins. The classical studies of Whipple and associates, as well as those of Rose, Daft and Sebrell and many others, have established the essential importance of the proteins to hematopoiesis; the globin demand of the hemoglobin molecule in experimental plasmaphoresis depleted dogs takes precedence over the needs of the body for plasma protein replacements. The complete nutritional requirements of the normal human individual are complex and numerous. Only those most likely to be overlooked in the day to day "stock piling," and which have highly specialized functional specificity, have been cited by example here.

Why killed vaccines are believed to produce a more effective immunity when injected than intrinsic foci of the same type of organism in the host is not clear, if they do. Reinfection, whether spontaneous, or induced in our monkeys, always produced evidence of specific circulating antibodies of a greater or lesser titer, depending upon the individual animal, and an effective cellular defense, unless nutritional deficiency or superimposed influenza virus infection intervened.

The R. E. system of phagocytic cells we believe to be largely, if not wholly, responsible for the humoral antibody specificity which follows streptococcus infection. The evidence of Sabin, and later of Houghton, would seem to point toward these cells as the source of the specific antibody globulins, rather than

the lymphocyte or plasma cell. The neutrophilic granulocyte is a short-lived "microphage" in *in vitro* tissue culture, and apparently *in vivo*, which provides instantaneous mobilization and attack, but which is easily destroyed during the battle. The other blood and connective tissue cells survive much longer and the increased cytoplasmic fragmentation of the highly phagocytic clasmatocyte on the ninth to thirteenth day following stimulation may be the source of the specific antibodies appearing about that time in the plasma globulins.

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